The Journal of the American Medical Association

Published Under the Auspices of the Board of Trustees

VOL. 129, NO. 13 — CHICAGO, ILLINOIS — NOVEMBER 24, 1945

COPYRIGHT, 1945, BY AMERICAN MEDICAL ASSOCIATION

PENICILLIN IN THE TREATMENT OF SUBACUTE BACTERIAL ENDOCARDITIS

A PRELIMINARY REPORT ON TWENTY CASES TREATED OVER ONE YEAR AGO

HARRISON F. FLIPPIN, M.D.
ROBERT L. MAYOCK, M.D.
FRANKLIN D. MURPHY, M.D.
AND
CHARLES C. WOLFERTH, M.D.
WITH THE TECHNICAL ASSISTANCE OF MARJORIE WILEY
PHILADELPHIA

In June 1942 an intern at the Philadelphia General Hospital suffering with subacute bacterial endocarditis was treated unsuccessfully by us with penicillin. The total dosage was 225,000 units given over a period of seven days. Similar results with relatively small dosage were observed by others. Subsequent to these early reports, Loewe and his associates [1] and Dawson and Hobby [2] were successful in the treatment of this disease by employing larger amounts of penicillin over longer periods of time. Following these encouraging results the Committee on Medical Research began a more extensive study of the use of penicillin in this disease, and this report represents a part of that investigation.

MATERIAL

This paper deals with the use of penicillin in the treatment of 20 cases [3] of subacute bacterial endocarditis in which therapy was started over one year ago. Not included in this report are 2 moribund patients, 1 of whom died following a cerebral thrombosis and the other as a result of a massive pulmonary hemorrhage after receiving the drug for only three and five days respectively. A third patient, who was not accepted for treatment because the infecting organism was highly resistant to penicillin, is also omitted.

CLINICAL DATA

The relevant data are shown in the accompanying table. Seventeen of the patients were adults ranging in age from 18 to 63 years, and the remaining 3 were children of 7, 10 and 12 respectively. Although it was impossible in many cases to determine the exact date of onset, the duration of disease at the time penicillin therapy was started has been approximated and found to vary from four to twenty-five weeks. Sixteen of the patients had rheumatic heart disease, 3 had congenital heart disease and the remaining 1 had arteriosclerotic heart disease. All of the patients in this study were of the white race.

INFECTING ORGANISM

Sensitivity of the infecting organism to penicillin was tested in all patients before treatment and was rechecked in those suffering relapses. The method used was similar to that described by Kolmer.[4] Fifteen of the patients' organisms showed an original sensitivity to 0.025 unit and the remaining 5 cases were sensitive to 0.01 unit of penicillin. In patients that relapsed the organisms showed a definite loss of sensitivity to penicillin. Sensitivity studies were made with penicillin X in 1 case (15).[5]

TREATMENT

For the most part the method of administering penicillin, the dosage and the length of treatment in the present series were quite uniform. Patients were given, if possible, an initial course of 300,000 units of penicillin a day for fourteen days by continuous intravenous infusion. In cases with severe cardiac decompensation and in those in which suitable veins were not available the same amount of penicillin was administered intramuscularly. Patients in moderate cardiac failure tolerated the 3,000 cc. of intravenous fluid daily, if the solution was 5 per cent glucose in water and the dietary salt was kept low. As indicated in the table, the dosage and duration of therapy varied in patients requiring further treatment. No heparin was employed in the treatment of any of these cases.

RESULTS

Of the 20 patients included in this report, 16 showed a subsidence of infection; 12 of these are still alive and, with only 1 exception (18), are all well. The follow-up period in these cases varied from two to seventeen months. In this group of living patients 10 received only a single course (4,200,000 units over fourteen days) of penicillin, while the remaining 2 (15 and 18) suffered relapses and required additional treatment. One of these patients (15) received a total of over 86,000,000 units,[6] administered intermittently during a period of almost twelve months, at the end of which

From the Harrison Department of Surgical Research, University of Pennsylvania School of Medicine, and the Medical Clinic, Hospital of the University of Pennsylvania.

The work described in this paper was done under contract, recommended by the Committee on Medical Research, between the Office of Scientific Research and Development and the University of Pennsylvania.

1. Loewe, Leo; Rosenblatt, Philip; Greene, H. J., and Russell, Mortimer: Combined Penicillin and Heparin Therapy of Subacute Bacterial Endocarditis: Report of 7 Consecutive Successfully Treated Patients, J. A. M. A. **124**: 144 (Jan. 15) 1944.

2. Dawson, M. H., and Hobby, G. L.: The Clinical Use of Penicillin: Observations in 100 Cases, J. A. M. A. **124**: 611 (March 4) 1944.

3. All of the patients were treated at the Hospital of the University of Pennsylvania except 1, who received treatment at the Philadelphia General Hospital.

4. Kolmer, J. A.: Penicillin Therapy, New York, D. Appleton-Century Company, Inc., 1945, p. 77.

5. Welch, Henry; Putnam, L. E.; Randall, W. A., and Herwick, R. P.: Penicillin X: Successful Treatment of Gonorrhea with a Single Intramuscular Injection, J. A. M. A. **126**: 1024 (Dec. 16) 1944.

6. The Donner Fund of the Hospital of the University of Pennsylvania made possible the continuation of penicillin therapy for several months in this case.

time the blood culture was still positive and the organism had become quite resistant to penicillin. This patient's organism was tested against penicillin X and found to be sensitive to 0.05 unit per cubic centimeter. The patient has been well for two months following the administration of 15,000,000 units of penicillin X over twenty-five days. The second patient (18) suffered three relapses, but since receiving 4,200,000 units intramuscularly over a period of twenty-one days she has been free from symptoms of the disease for over nine months. Within the past several weeks, however, she patient (20) died suddenly with an acute coronary occlusion after being entirely free from symptoms and signs of infection for almost a year. These 4 patients all died outside the hospital and no postmortem studies were performed. Two patients (1 and 19) failed to respond to penicillin and died in the hospital. At postmortem they were found to have active infection. Another patient (9) appeared to have responded to treatment only to die suddenly ten days later with what appeared to be an acute pulmonary embolism. The eighth patient who died (13) had two courses of

Analysis of Data on Twenty Cases of Subacute Bacterial Endocarditis Treated with Penicillin

No.	Age	Sex	Approximate Duration of Disease, Weeks	Primary Heart Disease	Infecting Type	Organism Penicillin Sensitivity, Unit/Cc.	Penicillin Therapy: Dates	Dosage, Units	Therapy Route	No. of Days	Result: Infection Arrested	Present Status	Comment	Follow-Up, Months
1	39	♀	12	Rheumatic	Streptococcus viridans	0.025	1/19/44	4,200,000	Intravenous	14	No	Died 4/9/44	Autopsy: vegetations on aortic and mitral valves	
						0.05	3/20/44	2,800,000	Intramuscular	14				
								7,000,000		28				
2	35	♂	10	Rheumatic	Strep. vir.	0.025	1/24/44	4,200,000	Intravenous	14	Yes	Died 7/1/44	Cardiac failure; no autopsy	
3	51	♂	6	Rheumatic	Strep. vir.	0.025	1/27/44	4,200,000	Intravenous	14	Yes	Living	Well	17
4	26	♀	22	Rheumatic	Strep. vir.	0.01	2/28/44	4,200,000	Intravenous	14	Yes	Died 9/24/44	Cardiac failure; negative blood culture; no autopsy	
5	18	♀	14	Congenital	Strep. vir.	0.025	2/28/44	4,200,000	Intravenous	14	Yes	Living	Well	16
6	41	♂	12	Rheumatic	Strep. vir.	0.025	3/ 2/44	4,200,000	Intravenous	14	Yes	Living	Well	16
7	44	♂	10	Rheumatic	Strep. vir.	0.025	3/13/44	4,200,000	Intravenous	14	Yes	Living	Well	15
8	10	♀	6	Congenital	Strep. vir.	0.025	3/26/44	4,200,000	Intravenous	14	Yes	Living	Well	15
9	23	♀	16	Rheumatic	Strep. vir.	0.025	3/31/44	2,000,000	Intravenous	7	?	Died 4/24/44	Probable pulmonary embolus; no autopsy	
							4/ 7/44	2,100,000	Intramuscular	7				
								4,200,000		14				
10	39	♀	12	Rheumatic	Strep. vir.	0.025	4/13/44	4,200,000	Intravenous	14	Yes	Living	Well	14
11	46	♀	25	Rheumatic	Strep. vir.	0.01	4/14/44	4,200,000	Intravenous	14	Yes	Died 2/24/45	Cardiac failure; negative blood culture; no autopsy	
						0.05	5/20/44	4,200,000	Intravenous	14				
							6/16/44	1,800,000	Sternal inf.	6				
							6/22/44	8,000,000	Intramuscular	90				
								18,200,000		124				
12	12	♀	16	Rheumatic	Strep. vir.	0.01	4/21/44	4,200,000	Intravenous	14	Yes	Living	Well	14
13	33	♂	10	Rheumatic	Strep. vir.	0.025	4/21/44	4,200,000	Intravenous	14	No	Died 3/7/45	Autopsy: Pseudomonas fluorescens endocarditis engrafted on subacute bacterial endocarditis	
							6/ 7/44	4,200,000	Intravenous	14				
						0.05	1/20/45	7,200,000	Intravenous	21				
								15,600,000		49				
14	38	♂	19	Rheumatic	Strep. vir.	0.025	5/11/44	4,200,000	Intravenous	14	Yes	Living	Well	14
15	26	♂	7	Rheumatic	Strep. vir.	0.025	5/16/44	4,200,000	Intravenous	14				
						0.05	7/10/44	4,200,000	Intravenous	14	Yes	Living	Well	2
							8/24/44	3,900,000	Intramuscular	39				
						0.5	10/ 2/44	8,600,000	Intramuscular	43				
						0.75	11/15/44	50,100,000	Intramuscular	167				
						0.05*	4/30/44	15,000,000*	Intravenous	25				
								86,000,000		302				
16	7	♂	12	Congenital	Strep. vir.	0.01	5/17/44	4,200,000	Intravenous	14	Yes	Living	Well	13
17	19	♀	10	Rheumatic	Indifferent streptococci	0.025	5/25/44	4,200,000	Intravenous	14	Yes	Living	Well	13
18	48	♀	8	Rheumatic	Strep. vir.	0.025	5/30/44	4,200,000	Intravenous	21				
						0.05	7/31/44	4,200,000	Intravenous	14	Yes	Living	Early cardiac failure	9
							8/20/44	5,600,000	Intramuscular	28				
							10/ 8/44	4,200,000	Intramuscular	21				
								18,200,000		84				
19	65	♀	4	Arterio-sclerotic	Indifferent streptococci	0.01	6/14/44	4,200,000	Intravenous	14				
							7/ 3/44	1,500,000	Intravenous	5	No	Died 7/23/44	Autopsy: vegetations on aortic valve	
							7/ 8/44	4,500,000	Intramuscular	9				
								10,200,000		28				
20	63	♂	8	Rheumatic	Strep. vir.	0.025	7/10/44	4,200,000	Intravenous	14	Yes	Died 5/27/44	Acute coronary occlusion; no autopsy	

* Penicillin X.

has developed signs of early cardiac failure with no evidence of active infection.

Eight of the patients in this study are dead (40 per cent). In this subgroup 3 (2, 4 and 11) died as a result of cardiac failure occurring five, six and three months after treatment. As indicated in the table, 1 of these (11) suffered relapses after receiving three courses of 300,000 units per day for fourteen days but apparently had a clinical subsidence of infection after receiving 50,000 to 100,000 units per day intramuscularly for three months. This patient died three months after therapy was completed from congestive failure with no signs of recurrence of infection. A fourth therapy, developed a secondary pseudomonas infection and died during the third course of penicillin. In this case thirty cultures were taken at appropriate intervals for six months after the second course and were negative, despite the presence of a continuous fever.

As already mentioned, there were 6 patients (1, 11, 13, 15, 18 and 19) who relapsed clinically and in each the blood culture was found positive. The time for recurrence of fever averaged twelve days, the longest being thirty days after treatment was finished. Blood cultures were always positive two to three days later, except in 1 case (13), in which the interval was six months. In this group of relapsed cases there was

observed a definite loss of sensitivity of the organism to penicillin. The culture in 1 case (19) was unfortunately lost. The most striking example of acquired resistance occurred in a man (patient 13) who received penicillin for almost a year. In this case the organisms showed an original sensitivity to 0.025 unit, which increased to 0.75 unit. The same organism was tested against penicillin X and found to be sensitive to 0.05 unit.

ERADICATION OF POSSIBLE FOCI OF INFECTION.

Since certain focal diseases may cause a recurrence of a case of subacute bacterial endocarditis, it is important that a careful search be made for possible foci of infection and their eradication effected if possible. Probably the most frequent focal diseases liberating bacteria into the circulating blood are tonsillitis, abscess of the teeth and otitis media. Because of the possibility of a transient bacteremia following tonsillectomy, dental extraction or the like we routinely administer sulfonamides or penicillin for several days before and after a surgical procedure. Furthermore, the patients are forewarned of the dangers of all dental manipulations and are advised to consult us about prophylactic measures if they contemplate having any dental work performed. Not included in this report is a patient with rheumatic heart disease who had a positive Streptococcus viridans blood stream infection and recovered following penicillin and the surgical removal of an arteriovenous aneurysm.

SUMMARY AND CONCLUSIONS

Twenty cases of subacute bacterial endocarditis were treated over one year ago with penicillin. Since there have recently appeared several excellent papers in which the salient features of this disease have been discussed, we will limit ourselves to several factors concerned with the management of these cases.

1. *Results of Penicillin Therapy.*—Of the 20 patients with subacute bacterial endocarditis, 16 had definite subsidence of their infection, 12 are still alive and with 1 exception are all well. Of the 8 who did not survive 4 were completely free of active infection and died as a result of heart disease. Three patients in this group failed to respond to therapy, and the eighth died soon after treatment was completed as a result of an acute pulmonary embolus.

2. *Establishment of Diagnosis by Positive Blood Cultures Before Starting Penicillin.*—Not infrequently a patient suffering with this disease is started on relatively small doses of penicillin before a positive blood culture is obtained. The dangers from this practice are threefold: 1. The organism may lose its sensitivity to penicillin. 2. The chances of obtaining a positive blood culture are lessened, despite the addition of penicillinase to the culture material. 3. The cardiac lesion itself undoubtedly progresses. Therefore it would seem best to withhold penicillin until a positive culture is obtained or, if the clinical diagnosis warrants, to administer adequate amounts of penicillin for the disease. In several cases, not included in this series, in which routine blood cultures were negative, we obtained positive results by employing anaerobic and arterial cultures.

3. *Adequate Penicillin Dosage.*—At this writing we are unable to make any conclusive statement regarding the amount or route of administration of penicillin for the treatment of this disease. As indicated, we employed 300,000 units a day for fourteen days as the initial course. This appeared adequate in 14 of the 20 cases. However, in view of subsequent experience with 16 additional cases of the disease, not included in this report, we believe that all patients with subacute bacterial endocarditis should receive at least 500,000 units a day for at least five weeks.

We have used a continuous intravenous infusion of penicillin where possible, but we have no reason to believe that this method is any better than the intermittent intramuscular route. Sternal infusion of penicillin is practical for limited periods of time.

4. *Eradication of Possible Foci of Infection.*—As a precaution against reinfection, a careful search for possible foci of infection, such as infected teeth and tonsils, should be made, and, if surgical procedure is warranted, the patients are given full doses of sulfonamides or, preferably, penicillin before and after operation.

5. *Penicillin X.*—In a case in which the infecting organism had become highly resistant to penicillin, we employed penicillin X with apparent success.

RHEUMATOID SPONDYLITIS

A STUDY OF ONE HUNDRED CASES, WITH SPECIAL REFERENCE TO DIAGNOSTIC CRITERIA

MAJOR EDWARD W. BOLAND
AND
MAJOR ARTHUR J. PRESENT
MEDICAL CORPS, ARMY OF THE UNITED STATES

Although rheumatoid spondylitis is one of the most crippling of the chronic diseases affecting the spine, it has evoked but little interest except among rheumatologists and orthopedists. Contrary to general belief, the disease is a fairly common cause of chronic back complaints in young men. Often the diagnosis is not established for several years after the onset, and the early symptoms are frequently mislabeled as lumbago, fibrositis or muscular rheumatism, chronic low back strain, kidney disease or idiopathic sciatica. If physicians are alert, however, to the characteristic clinical and roentgenographic features of the disease, diagnosis should not be difficult even in the early stage.

A surprisingly large number of soldiers with chronic back disability suffer from rheumatoid spondylitis; 18 per cent of patients with chronic back complaints admitted to an army general hospital were found to have this disease. The majority of these were early or relatively early cases. Such a high incidence among young men makes an appreciation of the diagnostic features, especially the early manifestations, important to physicians.

NATURE OF THE DISEASE

Rheumatoid spondylitis is a chronic progressive disease of the spine the fundamental lesions of which occur in the diarthrodial or synovial joints; that is, the sacroiliac, small posterior intervertebral (joints of the articulating processes, apophysial or facet joints) and costovertebral articulations. The intervertebral disks are not affected, nor are the vertebral bodies except for secondary demineralization. Calcific and later osseous changes develop in the paravertebral ligaments and, when pronounced, characterize the classic roentgenographic picture of a "bamboo spine"; but ligamentous

From the Medical and Radiologic Services, Hoff General Hospital, Santa Barbara, Calif.
Major Boland is now stationed at the Army and Navy General Hospital, Hot Springs National Park, Arkansas.

calcification is not an essential feature. The pathologic process usually, but not invariably, begins in the sacroiliac joints and then may spread upward to involve progressively the synovial joints of the lumbar, thoracic and cervical regions. With continued progression, movement of the spine decreases, a thoracic stoop develops, the chest becomes relatively fixed in expiration and the neck may protrude, resulting in the clinical picture of a "poker spine." The process may become arrested at any point, however, and such extensive deformity need not always eventuate.

Whether the disease is but an expression of rheumatoid arthritis as it involves the spine or a separate pathologic entity remains disputed. The contention that it is an independent disease is based on the following facts: (1) The prominent ligamentous calcification and ossification, seen in the later stages of spondylitis, is not a feature of rheumatoid arthritis involving peripheral joints; (2) the sex incidence in spondylitis favors males, while peripheral rheumatoid spondylitis is more common in females; (3) chrysotherapy may produce remissions in some cases of peripheral rheumatoid arthritis but is ineffective in spondylitis; (4) roentgen therapy may be of value in spondylitis but does not alter the course of rheumatoid arthritis affecting the peripheral joints. Those who believe that the disease is but rheumatoid arthritis of the spine point out that (1) typical rheumatoid arthritis involving peripheral joints frequently coexists, (2) the sedimentation rate is elevated and (3) the pathologic changes in the diarthrodial joints of the spine during active phases resemble those found in rheumatoid arthritis.[1] We believe, as do many other American investgatiors,[2] that the evidence favors the concept that the disease is a variant of rheumatoid arthritis.

A myriad of synonyms for rheumatoid spondylitis have appeared in the literature, based on (1) concepts of the nature of the process, (2) variations in location of involvement and (3) whether the disease was acute or chronic, transient or progressive. Such confusing terms as rhizomelic spondylitis, Marie Strümpell's disease, spondylitis ossificans ligamentosa of Knaggs, von Bechterew's syndrome, ankylosing spondylitis, adolescent or juvenile spondylitis, spondylitis ankylopoietica and spondylarthritis have been offered. The lack of uniform terminology has fostered confusion regarding the disease among physicians.

MATERIAL AND STUDY

One hundred soldiers with rheumatoid spondylitis were selected for study. No case was included in which the diagnosis was equivocal; roentgenographic findings in the sacroiliac joints, characteristic of the disease, were present in each case. Clinical and x-ray investigations and their correlation were made while the patients were under observation in the hospital.

The cases were classified on the basis of severity as determined by (1) the rapidity of clinical progression, (2) the degree of disability as manifested by the presenting symptoms and physical findings, (3) the amount of constitutional reaction, the height of the sedimentation rate and the degree of anemia and (4) the character of the roentgenographic changes. Such classification is more practical than a division based on roentgenographic findings alone, as has been suggested by some.[3] Roentgenographic manifestations usually fail to reveal the actual extension of the process as noted clinically and, although often helpful in gaging activity, they do not accurately reflect the clinical course of the disease. Of the 100 cases studied 40 were classified as mild, 50 as moderate and 10 as severe.

INCIDENCE, AGE AND SEX

Of patients with complaints referable to the musculoskeletal system due to a variety of causes, including arthritis, admitted to the medical service, 7.5 per cent were found to have rheumatoid spondylitis. One patient with spondylitis was admitted for every 3 with rheumatoid arthritis involving peripheral joints alone. These figures are in sharp contrast to those based on civilian practice. For example, 26 cases among 1,179 patients with arthritis were recognized by Hare,[4] an incidence of 1.7 per cent. The ratio of spondylitis to peripheral rheumatoid arthritis has been reported as 1:11,[5] 1:13[6] and 1:19.[4] The relatively high incidence of the disease in soldiers may be explained by (1) the preponderance of males in the army of an age group for which the disease has a predilection and (2) the influence of physical activity and adverse living conditions incident to military service in bringing to light mild and early cases. It is not probable that military life in itself is a causative factor. Of interest is the fact that German writers noted an increased frequency of the disease among soldiers in World War I.[7]

All of the patients studied were males. Rheumatoid spondylitis is chiefly confined to the male sex, the ratio of males to females affected being approximately 20:1.[8]

Ninety-two per cent of our cases were observed during the third and fourth decades of life. The onset of symptoms occurred between the ages of 20 and 29 years in 69 patients, between the ages of 30 and 39 years in 16 and between the ages of 16 and 19 years in 15. These figures are consistent with the findings of others.[9]

ASSOCIATED PERIPHERAL RHEUMATOID ARTHRITIS

Typical rheumatoid arthritis of the peripheral joints coexisted in 18 of the 100 cases; in 5 others a past history of joint disability accompanied by swelling was elicited, which from description suggested rheumatoid arthritis. In 8 of the 18 cases the peripheral joints were affected first, in 6 the first symptoms were in the back, and in 4 the involvement was more or less simultaneous. The hips were involved in 3 patients, the knees in 10, the ankles in 4, the metatarsophalangeal joints in 10, the proximal interphalangeal joints of the toes in 2, the shoulders in 3, the elbows in 3, the wrists in 5, the metacarpophalangeal joints in 6 and the proximal interphalangeal joints of the fingers in 4. These

1. Flynn, J. E.: Personal communication to the authors; unpublished data. Guntz, E., cited by Dunham and Kautz.[5]
2. Smyth, C. J.; Freyberg, R. H., and Lampe, Isadore: Roentgen Therapy for Rheumatoid Arthritis of the Spine (Marie-Strümpell Arthritis; Spondylitis Rhizomélique), J. A. M. A. **117:** 826-831 (Sept. 6) 1941. Miller, J. L.: Differential Diagnosis Between Strümpell-Marie Disease and Osteoarthritis of the Spine, J. Lab. & Clin. Med. **22:** 19-22, 1936. Hench, P. S.; Bauer, W.; Boland, E. W.; Dawson, M. H.; Freyberg, R. H.; Holbrook, W. P.; Key, J. A.; Lockie, L. M., and McEwen, C.: Rheumatism and Arthritis: Review of American and English Literature for 1940, Ann. Int. Med. **15:** 1002-1108, 1941. Oppenheimer, A.: Diseases of Apophysial (Intervertebral) Articulations, J. Bone & Joint Surg. **20:** 285-313, 1938.
3. Oppenheimer, A.: Development, Clinical Manifestations and Treatment of Rheumatoid Arthritis of the Apophysial Intervertebral Joints, Am. J. Roentgenol. **49:** 49-76, 1943. Scott, S. G.: Adolescent Spondylitis or Ankylosing Spondylitis, London, Oxford University Press, 1941. Forestier.[14]
4. Hare, H. F.: Diagnosis of Marie-Strümpell Arthritis, with Certain Aspects of Treatment, New England J. Med. **223:** 702-705, 1940.
5. Dunham, C. L., and Kautz, F. G.: Spondylarthritis Ankylopoietica: Review and Report of 20 Cases, Am. J. M. Sc. **201:** 232-250, 1941.
6. Tyson, T. L.: Spondylitis Ankylopoietica, M. Clin. North America **21:** 1755-1761, 1937.
7. Miller, J. L.: Chronic Rheumatic Diseases of the Spine, Arch. Int. Med. **54:** 161-169 (Aug.) 1934.
8. Bauer, W.: The Diagnosis of the Various Arthritides, New England J. Med. **221:** 524-533, 1939.
9. Buckley, C. W.: The Differential Diagnosis of Spinal Arthritis in Young Subjects, Brit. M. J. **2:** 4-6, 1943. Holbrook, W. P.; Stecher, R. M., and Hayden, E. M.: Primary Spondylitis, Ann. Int. Med. **6:** 1327-1334, 1933. Dunham and Kautz.[5] Hare.[4]

findings do not support the opinion that small peripheral joints are rarely involved.[10] Although the clinical and roentgenographic manifestations in the spine may be identical, some authors would classify those cases with associated small joint involvement as "rheumatoid spondylitis" and those without peripheral arthritis as "rhizomelic spondylitis." Such a division does not seem to be justified. The frequent association of rheumatoid arthritis of the extremities with the spinal changes of so-called rhizomelic spondylitis strongly supports the contention that the latter condition is rheumatoid arthritis of the spine.

PRECIPITATING FACTORS

Such possible precipitating factors as trauma, exposure, focal and other infections, psychic strain, gonorrhea and preceding subnormal health were studied in detail in each case. In 80 per cent no possible immediate precipitating cause could be elicited, and in the remaining 20 per cent no constant factor could be found. Eight patients dated their first symptoms to minor back trauma, but it is probable that in these injury merely brought an existing condition to light symptomatically. Four others had previously sustained incapacitating back injuries which were temporally unrelated to the onset of spondylitis. In 5 of the 100 cases acute gonorrheal urethritis was related to the onset of back symptoms, and in 8 others an unrelated past history of gonorrhea was elicited. The incidence of gonorrheal infection was approximately the same as that noted in patients with peripheral rheumatoid arthritis. The opinion expressed by some that gonorrhea may serve as an etiologic agent was not supported by the present study. Gonorrhea probably serves as a "trigger" mechanism (as may other infections) in some cases as it may in peripheral rheumatoid arthritis. In 1 instance a nonspecific urinary infection immediately preceded the first back symptoms. There was no definite relationship of dental, tonsillar or paranasal sinus foci of infection to the onset of spondylitis in any patient.

SO-CALLED PRESPONDYLITIC STAGE

Each patient was questioned in detail in an attempt to elicit and to evaluate peripheral muscle and joint complaints occurring prior to the onset of spinal symptoms. Scott[12] and others[13] described a "prespondylitic stage" characterized by wandering fleeting pains in the shoulders, arms, ribs and legs during which the sacroiliac joints presumably were silently involved. They maintained that spinal symptoms did not develop until bony ankylosis of the sacroiliac articulations was nearly complete. The present study yielded no evidence to justify the concept of a so-called prespondylitic stage. In 10 patients we were able to elicit a history of migratory peripheral joint and muscle complaints with or without stiffness. Such complaints usually were not impressive, or they had characteristics which suggested the syndrome of "fibrositis." Except for those patients with, or a past history of, rheumatoid arthritis involving the peripheral joints the first definite rheumatic complaints were almost always located in the back.

10. Buckley, C. W.: Ankylosing Spondylitis: Reports on Chronic Rheumatic Diseases, No. 1, New York, Macmillan Company, 1936. Herrick, W. W., and Tyson, T. L.: The Medical Aspect of Ankylosing Spondylitis (Marie-Strümpell), Ann. Int. Med. **15**: 994-1001, 1941. Golding.[13] Buckley.[9]

11. Footnote deleted on proof.

12. Scott, S. G.: Spondylitis Adolescens with Associated Pathologic Changes in the Sacroiliac Joints, Charterhouse Rheumat. Clin., Orig. Papers **1**: 169-203, 1937. Scott.[8]

13. Golding, F. C.: Spondylitis Ankylopoietica, Brit. J. Surg. **22**: 484-500, 1936. Hare.[4]

CLINICAL PICTURE

Mode of Onset.—In 82 of 100 cases the onset was insidious. Almost invariably the first complaints were referred to the lower part of the back and in 84 per cent consisted of episodes of aching and stiffness, transient back pain or sciatica.

Episodes of aching and stiffness, lasting a few days at a time and then subsiding completely or almost completely, were the most frequent initial complaints. Occasionally these symptoms were persistent from the onset. Qualitatively the aching and stiffness presented characteristics of the syndrome of "fibrositis," that is, being most pronounced in the morning on arising, accentuated by physical inactivity and ameliorated by mild exercise, made worse by damp weather and temporarily relieved by local heat and by acetylsalicylic acid. Although the intensity varied, the symptoms were mild in most cases; but, in a few, disabling attacks often described as "lumbago" occurred. Later the aching and stiffness tended to be more persistent and to lose the typical characteristics of fibrositis.

Less frequently the first symptoms consisted of transient sharp pains or "catches" in the buttocks and hips and in the lumbosacral, sacral or lumbar regions. Often these were precipitated by twisting, jolting, bending, lifting, coughing or sneezing but at times occurred spontaneously. Later, if not at the onset, aching and stiffness accompanied such pain. As the disease progressed, pain tended to become a constant dull discomfort, often described as a "deep ache," a "tired feeling" or a "soreness" and aggravated by activity entailing weight bearing or movement of the back. Not infrequently the discomfort was pronounced at night.

In 8 patients the first symptom was sciatic pain. In 6 it was intermittent and in 4 it alternated from one side to the other. The sciatica associated with rheumatoid spondylitis is rarely severe, is seldom accompanied by neurologic findings and frequently alternates from side to side.

Miscellaneous types of onset were recorded in 16 cases. A vague "tired aching" in the lower part of the back, especially following walking or standing, was noted by 4. Thoracic girdle pains constituted the initial complaint in 3. Pain in the right costovertebral angle radiating into the inguinal region, simulating renal colic, was described by 1. Three patients recalled progressive limitation of back motion without actual discomfort. One patient, bedridden with severe peripheral rheumatoid arthritis, had noted only vague, ill defined backache. Three others, with long standing spondylitis, were unable to recall the initial symptoms.

Symptoms and Findings.—Although the overall pattern of rheumatoid spondylitis is fairly characteristic, the clinical picture at the time of examination is variable. The symptoms and findings are dependent on (1) the severity, rate of progression and duration of the disease, (2) the extent of spinal involvement and (3) the activity of the process at individual levels of the spine. The disease may be mild, moderate or severe; it may be localized to the sacroiliac joints or at the other extreme may involve the synovial joints of the entire spine; it may be semidormant and silent in one area while active and symptomatic in another. The severity of symptoms and the extent of the physical findings are dependent on such qualifying factors.

While the disease may begin at any level in the spine, it begins in the sacroiliac joints in the vast majority of instances. The process may subside temporarily or may terminate permanently at any point, but the ten-

dency is toward a gradual and relentless spread to higher levels. Hence in most instances the sacroiliac joints are involved at the time of examination, and, as a rule, the lumbar spine is affected before symptoms appear in the thoracic spine. Occasionally, however,

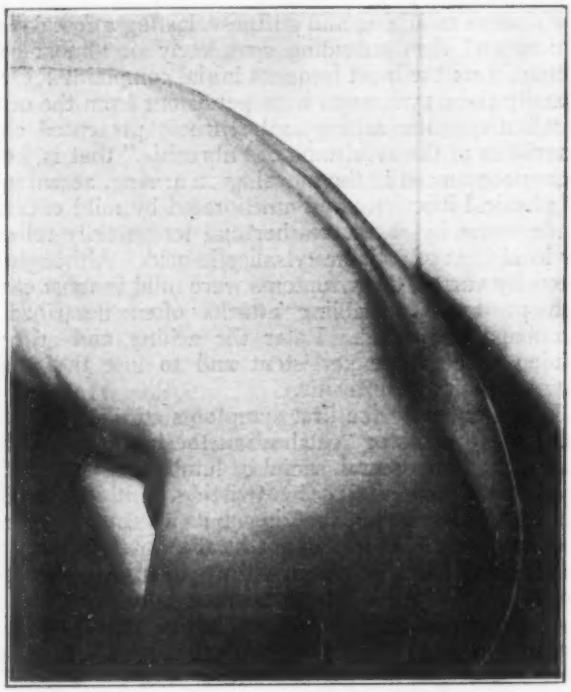


Fig. 1.—Contour of the normal back with the trunk moderately flexed. The contour may be normal when involvement is restricted to the sacroiliac joints.

Fig. 2.—Moderately advanced rheumatoid spondylitis, showing limitation of motion and straightening of lumbar spine. Paravertebral muscle spasm is present in the lower thoracic and upper lumbar regions. Note the muscle atrophy in the lumbosacral area ("ironed out" appearance).

the disease apparently by-passes one or even two segments and consequently different combinations of symptoms and findings may be encountered. As it would not be practical to describe all possible variations, an enumeration of the features resulting from involvement in individual segments may be useful.

Sacroiliac Involvement.—When these joints alone are the sites of active rheumatoid arthritis, the symptoms consist of aching, stiffness and pain in the lower part of the back which qualitatively have already been described. Sciatica occurs in approximately 10 per cent of instances. Tenderness over one or both sacroiliac joints may be elicited on percussion or deep palpation in over 50 per cent of patients with early changes; as ankylosis develops, tenderness (and pain) disappear. Various orthopedic maneuvers which produce sacroiliac joint movement may cause local pain. Mild lumbar paravertebral muscle spasm without restriction of lumbar motion is frequent; pronounced muscle spasm usually indicates lumbar apophysial joint involvement. In very mild cases or when the sacroiliac joints are ankylosed (and unaccompanied by higher involvement), physical signs may be entirely absent.

Fig. 3.—Characteristic posture in spondylitis involving entire spine. Note the lumbodorsal stoop, the flattened anterior chest and the protruded neck. Both hips are involved, with flexion deformity on the left.

Lumbar Involvement.—The lumbar segment is rarely involved alone, and almost invariably the disease has extended from the sacroiliac joints. Pain, aching and stiffness, as already noted, remain the dominant symptoms but are not of great localizing value. Sciatica is not uncommon. Among the signs which signify involvement of the lumbar spine are (1) limitation of motion of the lumbar spine, (2) paravertebral muscle spasm, (3) straightening of the normal lumbar lordosis, (4) tenderness (constant) to percussion and deep palpation over or just lateral to the lumbar spinous processes, (5) pain in the lumbar spine on flexion (anterior and lateral) and extension, (6) muscle atrophy, especially in the lower portion of the lumbar segment, which when combined with straightening gives the lower back an "ironed out" appearance (figs. 1 and 2), and (7) anterior stoop (usually associated with thoracic involvement).

Thoracic Involvement.—When the thoracic segment is affected the findings result from involvement of the thoracic posterior intervertebral joints, the costovertebral joints and the thoracic nerve roots. These include (1) tenderness (constant) on percussion and deep palpation over or just lateral to the thoracic spinous processes, (2) muscle spasm and later muscle atrophy,

(3) limitation of motion of the thoracic spine, (4) girdle pains, (5) diminished chest expansion with reduction of vital capacity, (6) flattened anterior chest (expiratory position, fig. 3), (7) chest pain on forced inspiration and (8) thoracic kyphosis (thoracolumbar stoop).

Cervical Involvement.—With involvement of this segment the features presented are (1) tenderness (constant) on percussion and palpation over the posterior neck, (2) pain (constant) on forced motion of the cervical spine, (3) restricted motion of the neck, (4) spasm and later atrophy of the cervical muscles, (5) protruded position of the neck and (6) cervical radicular pain (rare).

Constitutional Reaction.—General constitutional symptoms such as weakness, fatigue, anorexia, weight loss and low grade fever are rarely prominent features of spondylitis unless the disease is severe or unless peripheral joints are concomitantly involved. Such reaction, as a rule, parallels the height of the sedimentation rate. Mild cases may be accompanied by minimal or no constitutional symptoms, and many patients may

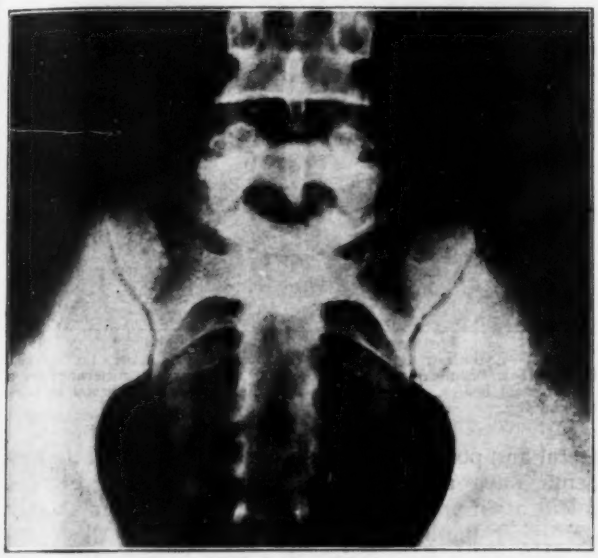


Fig. 4.—Normal sacroiliac joints. Note the clear joint spaces and the sharp borders.

pursue useful occupations for years without appreciable alteration in general health.

Laboratory Findings.—The erythrocyte sedimentation rate serves as the most consistent laboratory gage of activity in spondylitis, just as it does in peripheral rheumatoid arthritis. It was elevated in 78 of 100 cases. The usual range was between 20 and 60 mm. in one hour (modified Westergren, normal under 20 mm. in one hour for males), the most rapid being 112 mm. in one hour. In several patients with mild but active disease the sedimentation rate was repeatedly within normal limits. Others have observed that when the disease is mild with insidious onset and slow progression the sedimentation rate may be normal.[14] Moderate hypochromic anemia was present in 28 patients.

ROENTGENOGRAPHIC FEATURES

The roentgenographic features of rheumatoid spondylitis are quite characteristic. In general they are reflections of those pathologic changes which develop in the sacroiliac and apophysial articulations and in the paraspinal ligaments. Except for secondary demineralization the vertebral bodies are not affected. The intervertebral disks remain intact.

X-ray alterations of the synovial joints result from destruction of articular cartilage and changes in the subchondral bone. When the pathologic process is

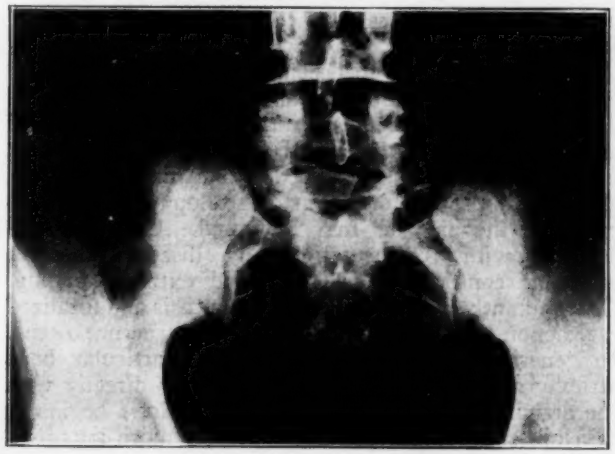


Fig. 5.—Moderately advanced mild rheumatoid spondylitis. The sacroiliac joints are blurred and the margins are indistinct. Note the juxta-articular increase in bone density in the ilium and sacrum.

restricted to the synovial membrane, the x-ray appearances are normal. Synovitis may exist for months or even for two to three years before cartilage or bone is sufficiently modified to be recorded by roentgenograms. Hence symptoms of the disease may be present long before positive x-ray diagnosis is possible. Similarly, in patients with ascending disease there is an appreciable lag between the development of physical findings and x-ray alterations.

Sacroiliac Joint Changes.—Significant alterations in the sacroiliac joints existed in each of the 100 patients; the involvement was bilateral in 96. Although rheumatoid spondylitis may be suspected on clinical grounds alone, we have hesitated to make an unequivocal diagnosis when the sacroiliac joints have appeared normal.

Fig. 6.—Moderately advanced rheumatoid spondylitis of moderate severity. The sacroiliac joints are indistinct and mottled. Note the serrated sacroiliac joint borders and the juxta-articular sclerosis.

Abnormalities in these joints almost invariably are more definite, more advanced and more diagnostically reliable than are alterations elsewhere in the spine.

The normal sacroiliac joint in young men is clear and its borders are sharply outlined. The earliest changes in spondylitis consist in a blurring or "ground"

14. Forestier, J.: Importance of Sacroiliac Changes in the Early Diagnosis of Ankylosing Spondylarthritis: Marie-Strümpell-Bechterew Disease, Radiology **33**: 289-402, 1939. Golding.[33]

glass appearance. The margins are no longer distinct or they may appear broken. The juxta-articular portion of the ilium and frequently the sacrum may demonstrate increased density, spotty osteoporosis or both.

Subsequent changes are at least partially dependent on the severity and the duration of the disease. When the process is mild, juxta-articular sclerosis is more prominent than spotty rarefaction, and mottling of the joint space is minimal or absent (figs. 4 and 5). Rather, the joint space tends to become less distinct and more narrowed until ankylosis finally develops. When the disease is more severe the joint space appears widened and irregularly mottled (fig. 6). The lower portion may assume a "honey combed" appearance, and later extensive "rosary" formation or scalloping with cystic caries of the margins may be found. Complete dissolution with fragmentation and disappearance of sacroiliac contours is an occasional extreme finding. Concomitantly, varying degrees of sclerosis and localized rarefaction in the adjacent ileum and sacrum occur. In general the degree of spotty juxta-articular bone atrophy and joint mottling seem to vary directly with the activity of the disease. When ankylosis begins to develop, subchondral sclerosis becomes more apparent, mottling is less pronounced and the joint space gradually becomes narrowed. When synosteosis is established, the areas of increased density fade. Eventually there is continuity between the sacrum and ilium with little or no trace of the joint (fig. 7).

Posterior Intervertebral (Apophysial) Joint Changes.—By no means were changes in the apophysial joints as definite or as constant as those in the sacroiliac articulations. Even with the use of special technics, detailed studies often yielded disappointing results. In no instance in this series were there typical findings in the apophysial joints without accompanying evidence of sacroiliitis. When apophysial changes were found usually only a few scattered joints were abnormal while intervening ones appeared normal. Presumably the pathologic process in the interposed normal appearing articulations had not produced sufficient cartilaginous or bony change to be registered roentgenographically.

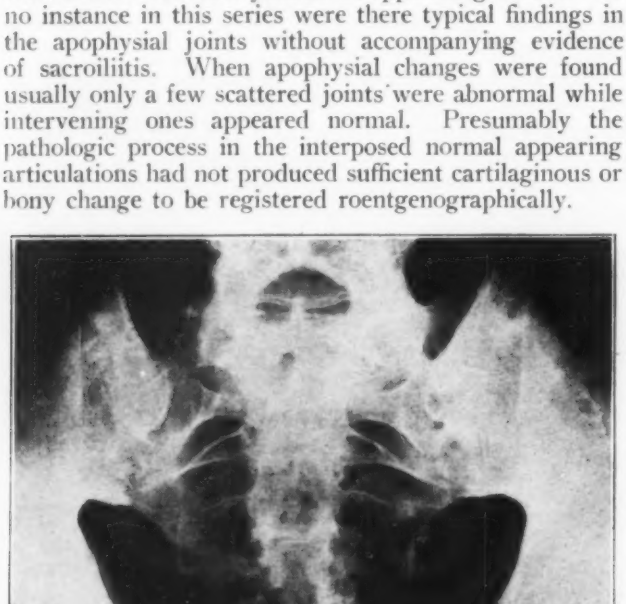


Fig. 7.—The sacroiliac joints are ankylosed. Note the demineralization, which is a common finding when fusion is complete.

Qualitatively, the apophysial alterations were similar to those found in the sacroiliac articulations. Early there were haziness and indistinct outlining of the joint. Stippled decalcification and/or sclerosis of the articular process was often evident. Later, in more severe cases, erosion, scalloping or perhaps a single cystic rarefaction of the facet surface was a typical finding. All degrees of destruction have been noted in the same spine with completely normal intervening joints at some levels. As in the sacroiliac joints, the final event consisted of bony ankylosis, but even in this phase not all the joints of an involved segment were fused.

Paraspinal Ligament Changes.—Calcification and ossification of the paravertebral ligaments do not develop until the disease is moderately advanced. The anterior,

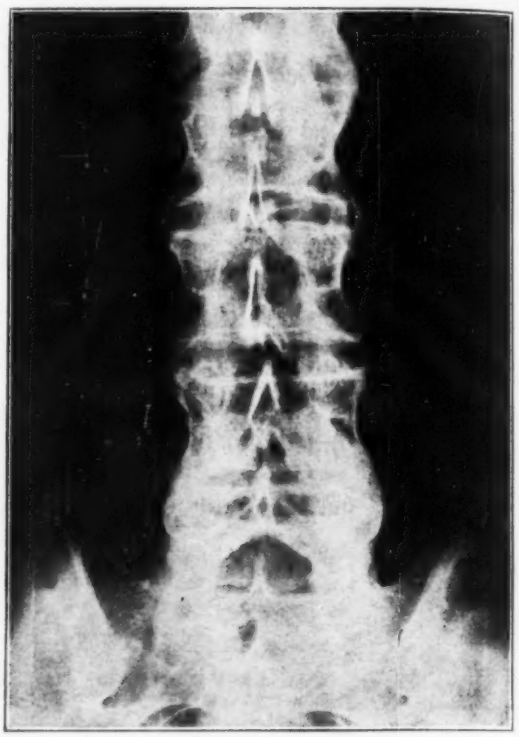


Fig. 8.—Extensive ligamentous calcification producing the terminal picture of a "bamboo spine." Note the secondary demineralization of the vertebral bodies. The intervertebral disk spaces are normal in height and contour.

lateral and posterior longitudinal ligaments and the ligamenta flava are the usual sites of calcific changes. Calcification most often begins in the lower thoracic and upper lumbar portions of the spine. All degrees of calcification and ossification may be found, ranging from fine penciled lines in the anterior spinal ligament to extensive "bambooing" (fig. 8). In a few exceptional cases definite ossification of the ligaments was observed in the lower thoracic and upper lumbar spine when only minimal and early changes were present in the sacroiliac articulations. In these instances the disease probably began in the apophysial joints of the thoracolumbar spine rather than the sacroiliac articulations.

CLINICAL COURSE

The course of rheumatoid spondylitis is extremely variable and to a large degree is dependent on the severity of the disease. Mild cases, which are characterized by insidious onset, little or no elevation of sedimentation rate, minimal or no constitutional symptoms and x-ray changes showing predominant juxta-articular sclerosis rather than spotty osteoporosis or joint mottling, tend to run a relatively benign course. Symptoms may be mild and not incapacitating for years. Progression of the disease is gradual but none the less relentless, and the stage of "poker back" deformity may not be reached for fifteen to twenty-five years. As has already been pointed out, the disease may, however, temporarily or permanently subside at any point. For example, it may terminate before it has extended beyond

the sacroiliac joints, and the only residue of the disease may be the x-ray "ashes" of completely ankylosed sacroiliac joints. To be kept in mind is the fact that an apparently "burned out" process may become reactivated at any time.

In moderately severe cases back disability is usually of sufficient severity for the patient to seek medical aid. Progression is more rapid, the sedimentation rate is almost invariably elevated and the roentgenograms reveal changes which signify a higher degree of disease activity. Pronounced restriction of back motion, diminished chest expansion, lumbodorsal stoop and neck involvement may be evident within five to ten years in those cases which show continued extension. The activity of the disease may fluctuate, however, and there may be alternating periods of temporary improvement and exacerbation.

Severe instances of the disease usually demonstrate pronounced back disability, definite constitutional reaction and rapid progression. We have observed extreme deformity with complete rigidity of the spine in all segments ten months after the first onset of symptoms. In some, spondylitis is merely a part of a severe generalized rheumatoid arthritis in which the peripheral joints share equally in the involvement.

DIAGNOSIS

When rheumatoid spondylitis has advanced sufficiently to produce characteristic physical and roentgenographic signs, little difficulty should be encountered in diagnosis. Such clinical features as progressive limitation of spinal motion, pronounced muscle spasm, straightening of the normal lumbar lordosis, muscle atrophy, lumbodorsal stoop, diminished chest excursion, flattening of the anterior thorax or protrusion of the neck, when observed in almost any combination, should serve as adequate clues. Such roentgenographic findings as bilateral sacroiliitis, apophysial joint changes and straightening of the lumbar spine, perhaps accompanied by ligamentous calcification and ossification, should confirm the diagnosis. Other features, such as an elevated sedimentation rate, constitutional symptoms or associated peripheral rheumatoid arthritis, serve as additional evidence. The extreme case with poker back deformity can be readily spotted on the street corner.

Early cases may be easily missed, however, unless the possibility of the disease is considered in every patient with chronic back complaints. The following points, axiomatically summarized, may be helpful in recognizing early rheumatoid spondylitis:

1. Suspect rheumatoid spondylitis when a young man complains of chronic recurrent or persistent low back aching and stiffness, with or without catching pains, especially if the sedimentation rate is elevated.

2. Suspect rheumatoid spondylitis in the young man who complains of such vague symptoms as a "tired feeling" in the lower part of the back on standing and walking, persistent low back soreness, silent restriction of back motion, or indefinite sharp pains in the buttocks, hips or lower part of the back, especially if accompanied by an elevated sedimentation rate or general constitutional symptoms.

3. Suspect rheumatoid spondylitis in all cases of sciatica in young men, particularly if recurrent or alternating from side to side or associated with aching and stiffness of the lower back.

4. Suspect rheumatoid spondylitis in patients with thoracic girdle pains, especially if accompanied by symptoms in the lower part of the back.

5. Suspect rheumatoid spondylitis when persistent back symptoms develop in a patient with peripheral rheumatoid arthritis.

6. In the absence of roentgenographic evidence of sacroiliac involvement an unequivocal diagnosis of rheumatoid spondylitis should not be made unless characteristic changes are present in the apophysial joints.

7. Remember that characteristic x-ray changes in the sacroiliac or apophysial joints may not develop for months after the onset of symptoms. Do not eliminate the possibility of rheumatoid spondylitis on negative x-rays alone unless persistent symptoms have existed for at least three years.

8. Definite bilateral destructive and/or sclerotic changes in the sacroiliac joints, noted roentgenographically, almost invariably indicate rheumatoid spondylitis.

9. Be cautious in making a diagnosis of rheumatoid spondylitis with unilateral sacroiliac involvement unless other characteristics of the disease are present or unless peripheral rheumatoid arthritis coexists. Persistent unilateral sacroiliitis may be due to tuberculosis.

10. Calcification of the paravertebral ligaments may result from several causes and in itself is not sufficient evidence for the diagnosis of rheumatoid spondylitis; changes in the apophysial and/or sacroiliac joints must also be present.

11. Remember that the sedimentation rate may be normal in 15 to 20 per cent of cases with active disease and that constitutional symptoms are usually milder than in rheumatoid arthritis involving peripheral joints.

EARLY RISING IN THE PUERPERIUM

GORDON ROSENBLUM, M.D.
EUGENE MELINKOFF, M.D.
AND
HARRY S. FIST, M.D.
LOS ANGELES

HISTORICAL BACKGROUND

The practice of early rising of parturient women dates back to ancient times. Although it was recently stimulated by the wartime shortage of obstetric beds, it did not arise from that emergency. The puerperium, or time of rest given to women in childbed, has varied more widely in the customs of different peoples than any other feature of that great physiologic function of woman.

The biblical Israelites considered the puerpera unclean for seven days after the birth of a male child and for fourteen days after the birth of a female child, during which period she was required to remain at home.[1] The longer period of isolation after a female child has been attributed to their belief that the female sex, being inferior, require a longer period to restore cleanliness. In Athens the puerpera was considered unclean, and whoever touched the mother was forbidden to visit an altar. There can be no doubt that these religious practices in and of themselves enforced a period of at least partial rest during the puerperium. However, the northern tribes of Russia, the Samojedes and others who considered the puerpera unclean often left her

From the Department of Obstetrics and Gynecology, Cedars of Lebanon Hospital.

Miss Goldie Stein of the Cedars of Lebanon Hospital record room assisted in collecting the records.

1. Rongy, A. J.: Childbirth: Yesterday and Today, New York, Emerson Books, Inc., 1937, p. 31. Kotelman: The Ancient Hebrews.

in such a state of isolation and neglect that both mother and child perished.[2]

In Egypt the wealthier class of women remained in bed three days post partum, but poor women usually resumed their occupations in one day. Syrian women were permitted six days of rest.

The Wakamba of Africa put their parturient women to work after a four day confinement. The North American Indians and other Indian tribes observed no period of puerperium, their women returning to work the day of delivery, following the ceremonial bath, which was often performed in the river.[3] In this connection Dr. J. Fields[4] wrote in 1883 "During two and a half years' life among the Indians I never saw or heard of a case of puerperal fever, puerperal eclampsia or any other diseases peculiar to lying-in women."

Sioux Indian squaws have been known to go into the woods in the cold months of winter and, while there, to give birth to the papoose, which they brought back, together with a load of wood to keep the home fires burning. According to Engelmann, the Apache Indians believed it essential that as soon as the placenta was expelled the parturient be kept upright and walking for a half hour in order that all clotted blood be expelled from the womb.[2] The same authority also stated that the Japanese, prior to the nineteenth century, shared in this belief concerning the upright position and kept their parturients propped up in a sitting position for three days before allowing them to recline.

In the Antilles and Guianas food was offered as soon as delivery was completed, and after a few hours the usual labor of the household was resumed by the patient. In old Calabar, in Greenland and among certain Canadian tribes the custom formerly practiced was to allow the delivered woman immediately to resume her household duties while the husband went to bed and received the visits of friends and relatives.[2]

The English obstetrician Charles White[5] of Manchester wrote in 1793 "The parturient should lie very high with her head and shoulders and should sit up in bed many times a day, especially when she takes food, and as often as she suckles her child, and she should kneel whenever she has occasion to make water, which should be done often. The frequent upright posture is of the utmost consequence and cannot be too much enforced. It prevents the lochia from stagnating, the stools and urine from being too long retained, and promotes the contraction of the uterus, together with that of the abdominal muscles."

In reference to early puerperal rising, Charles White also wrote "The sooner she gets out of bed after her delivery, the better; even on the same day if possible; she should not defer it beyond the second or third at the fartherest, and then if it be winter time it will be necessary to have a fire." From his writings it appears that Charles White believed early puerperal rising was a prophylaxis against puerperal fever.

Rotstein[6] asserted that it had been demonstrated that the early rising of puerperal women necessitated by the London blitz of 1940-1941 did not result in harm to the patient, and he advocated this method of puerperal management. Several other reports advocating early puerperal rising appear in American and foreign literature. More recently, Black[7] reported an increased obstetric bed turnover at the Alameda County Hospital, Oakland, Calif., by allowing his patients home early in the puerperium. In his reported series of 3,400 cases about one third were discharged on their fourth day, 30 per cent between the fourth and eighth days and 35 per cent after the eighth day. Although not clearly stated in Black's article, we may presume that most patients were ambulatory, at the time of discharge from the hospital. He reported that no complications resulted from this practice. Black concluded after a twelve year program, during which the hospital staff cared for 12,000 deliveries, that a short, active puerperium was safe and desirable. The patients in the entire series were allowed out of bed at varying intervals according to their condition.

Armand[8] claimed that, in a group of 200 puerperal women in Haiti, early rising prevents thrombosis and embolism, aids uterine involution, helps lochial drainage and fosters quick recovery. He did not observe a single case of inversion, prolapse or retroversion of the uterus. He strongly advocates early rising in the puerperium regardless of the type of delivery. Jorge[9] allowed only normal puerperal women to get up and reported evident benefit. Vara[10] writes that in the women's clinic at Helsingfors 4,447 women were allowed out of bed very soon after delivery, with resulting improvement in bowel and bladder function and less incidence of thrombosis than in those who were kept in bed for longer periods.

Greenhill[11] writes: "Beginning about twenty years ago I gradually cut down the number of days my obstetric and gynecologic patients remained in bed. Now I have progressed so far that, if my obstetric patients cannot urinate spontaneously eight to ten hours after delivery and are uncomfortable as a result of a full bladder, I have them helped out of bed either to sit on a commode or to go to the toilet if there is one in the room. Naturally, women who have bled a great deal or have had complications are not permitted to do this, but at least 90 per cent of parturient women could be permitted out of bed on the first day without harm. . . ." Nelson and Collins[12] reported good results in both obstetric and gynecologic cases in which cotton sutures, together with early ambulation, were employed.

The practice of early ambulation of surgical patients, now a widely accepted procedure, advocated by hundreds of articles in the American and foreign literature, must be considered intimately related to the practice of early puerperal rising. The objectives and underlying principles of the two are identical. Newberger, in a collective review,[13] presented 189 references advo-

2. Engelmann, G. J.: Labor Among Primitive Peoples, St. Louis, J. H. Chambers & Co., 1883.
3. Corlett, W. T.: The Medicine Man of the American Indian and His Cultural Background, Springfield, Ill., Charles C Thomas, Publisher, 1935.
4. Fields, J., cited by Engelmann: Labor Among Primitive Peoples, p. 32.
5. White, Charles: Treatise on the Management of Pregnant and Lying-in Women, 1793, Worcester, Mass., I. Thomas, 1793.
6. Rotstein, M. L.: Getting Patients Out of Bed Early in the Puerperium, J. A. M. A. **125**: 838-840 (July 22) 1944.
7. Black, B. W.: Do Normal Maternity Cases Require Ten Days in Hospital? Mod. Hosp. **60**: 52-53 (Feb.) 1943.
8. Armand, F. M.: Lever precoce dans les suites des couches, Obst. y ginec. latino-am. **1**: 688-697 (Dec.) 1943.
9. Ferreira Jorge, Cid: Levantar precoce em obstetricia e o serviço de assistência obstétrica a domicílio, Rev. de ginec. e d'obst. **2**: 288-291 (Nov.) 1943.
10. Vara, P.: Beobachtungen über das "Frühaufstehen" nach gynäkologischen Operationen bezw. Entbindungen, Acta obst. et gynec. Scandinav. **21**: 168-179, 1941.
11. Greenhill, J. P.: Year Book of Obstetrics and Gynecology, Chicago Year Book Publishers, Inc., 1944.
12. Nelson, E. W., and Collins, C. S.: Cotton Suture Material and Early Ambulation in Obstetrics and Gynecology, Surgery **12**: 109-114 (July) 1942.
13. Newberger, B.: Early Postoperative Walking, Surgery **13**: 692-695 (May) 1943.

cating early rising of surgical patients. He enumerates the following advantages gained from his study of the literature:

1. Asthenia is minimized.
2. The morale of the patient is lifted.
3. Economy to both patient and hospital is obtained through more rapid convalescence and through reduction of nursing personnel and more efficient use of bed space.
4. Military surgery may be rendered less hazardous.
5. Simplification of postoperative care is obtained.
6. Pulmonary complications are reduced four to five fold.
7. Hollow viscus atony is reduced, with consequent avoidance of use of catheters and laxatives.
8. Wound healing is improved.
9. Thrombosis and embolism are reduced.

Leithauser [14] reported excellent results in several hundred surgical patients who were got up during the first twenty-four hours following surgical operation and claimed that this treatment prevented most pulmonary and circulatory complications. He studied twenty-nine articles from the foreign literature, among which was the particularly impressive report of Zava [15] of 6,000 patients who were got out of bed on the first day without the occurrence of a single case of embolism or eventration. It is interesting to observe that Newberger [13] showed that the comparative healing of wounds was better in ambulated than in nonambulated rats.

Many observers, including Churchill and McNeile,[16] have noted a definite decrease in the minute volume respiratory exchange of postoperative patients. This decreased respiratory exchange results from such factors as the depression caused by preoperative medication, the use of depressant anesthetic agents and various other factors, such as muscle splinting. Beecher [17] established that this reduction is most pronounced in the first twenty-four hours. Hoerr [18] showed that 50 per cent of all primary postoperative pulmonary complications occur within the first twenty-four hours and that 90 per cent make their appearance before the end of the fourth day. Leithauser [14] reported that spirometer readings showed that vital capacity returned to normal much sooner after early rising.

One of us (G. R.) had advocated and practiced since 1940 the early ambulation of postoperative gynecologic patients. In a report to the obstetric and gynecologic staff of the Cedars of Lebanon Hospital in 1941 he also advised the use of the transverse abdominal incision in obstetrics and gynecology, pointing out that patients operated on in this manner could advantageously be made ambulatory within twenty-four hours. In patients in whom the transverse incisions were made there occurs less postoperative discomfort and there is a lower incidence of postoperative hernias and eviscerations. Consequently patients operated on in this manner can be more safely got up within the first day, as compared to those in whom the more conventional types of incisions are made. Accordingly, in the cesarean sections to be reported later in this paper the transverse abdominal incision was used except when a previous midline incision existed.

With this background of favorable evidence in the literature, the task of studying early ambulation in the puerperium was begun. At the outset it was realized that there would be objections both from patients and from the medical profession to the practice of early ambulation. The following objections arose during the course of our study:

Objections by Doctors.—1. Fear of medicolegal consequences. 2. Fear that episiotomies would break down. 3. Fear that there might be too much strain on the pelvic floor, resulting in prolapses and retroversions. 4. Fear of excessive postpartum bleeding. 5. Fear that patients would not approve of early rising.

Objections by Patients.—1. That the procedure was unfamiliar to them, therefore they were not sure it was good. 2. That they needed rest and it might tire them too much to get up. 3. That they were too weak to walk. 4. That arising might make them bleed too much. 5. That they might "break their stitches."

We believed that these objections were more theoretical than actual, therefore an attempt to evaluate them accurately was begun.

TABLE 1.—*Statistics*

Total number of cases in series				582	
Total cesarean sections				50	8.6%
Early risers				247	42.4%
Vaginal deliveries		229	92.7%		
Spontaneous	104				
Low forceps	107				
Mid forceps	4				
Breech extraction	10				
Version	1				
Twins	3				
Cesarean section		18	7.3%		
Intermediate risers				106	18.2%
Vaginal deliveries		103	91.2%		
Spontaneous	33				
Low forceps	65				
Mid forceps	3				
Breech extraction	0				
Version	0				
Twins	2				
Cesarean section		3	2.8%		
Late risers				229	39.3%
Vaginal deliveries		200	87.3%		
Spontaneous	85				
Low forceps	92				
Mid forceps	10				
Breech extraction	9				
Version	0				
Twins	4				
Cesarean section		29	12.7%		

METHOD OF STUDY

The articles, such as that of Black,[7] which have advocated mobilization of puerperal women in an intermediate period had led us to the division of our total group of 582 obstetric patients into three groups as follows:

1. Early risers: ambulatory on first or second postpartum day.
2. Intermediate risers: ambulatory on third or fourth postpartum day.
3. Late risers: ambulatory later than the fourth postpartum day.

A consecutive series of 229 unselected patients delivered by us prior to March 1944 was taken from the files in the record room. These patients had all been made ambulatory later than five days post partum. These are called "late risers" and serve as controls. Beginning with March 1944, 106 delivered women, unselected in any manner, were made ambulatory on the third or fourth postpartum days. These women, the "intermediate risers," were given the privilege of going to the bathroom, which was always in the room, instead of using bed pans. In addition, they could eat their meals out of bed if desired, leave the bed to bathe and remain up while the bed linen was being changed. However, the rest of the time they were

14. Leithauser, D. J.: Confinement After Operation, Arch. Surg. **42**: 1086-1093 (June) 1941; ibid. **47**: 203-214 (Aug.) 1943.
15. Zava, L.: Sui vantaggi del levar precoce degli operati, Policlinico (sez. prat.) **47**: 865-872 (May 20) 1940.
16. Churchill, E. D., and McNeile, D.: Reduction in Vital Capacity Following Operation, Surg., Gynec. & Obst. **44**: 483-488 (April) 1927.
17. Beecher, H. K.: Measured Effect of Laparotomy on Respiration, J. Clin. Investigation **12**: 639-650 (July) 1933.
18. Hoerr, S. O., and Culter, E. C.: Postoperative Pulmonary Complications, Proc. Interst. Postgrad. M. A. North America **9**: 232-237, 1942.

asked to remain in bed. Thus, the time actually spent in bed must have amounted to 95 per cent of the total time. The routine as regards postpartum care was otherwise not changed.

During the interval in which the "intermediate riser" group was being accumulated, we began to study an unselected series of women who were made ambulatory during their first or second postpartum days. This group, comprising 247 patients, is called the "early risers." [19] Aside from early ambulation, there were no changes made in the management of these patients. Patients delivered by cesarean section were included in this series without selection.

TABLE 2.—*Bladder Function*

	Early		Intermediate		Late	
	No.	%	No.	%	No.	%
Number in series	247		106		229	
Number studied *	246	100	104	100	229	100
Number of patients requiring catheterization	87	35.4	42	40.4	89	38.9
Number of patients not requiring catheterization	159	64.6	62	59.6	140	61.1
Total number of catheterizations †	266		171		341	
Average number of catheterizations per patient	3.0		4.0		3.8	
Voiding began on first postpartum day	240	97.6	96	92.3	221	96.5
Voiding did not begin on first postpartum day	6	2.4	8	7.7	8	3.5
Voiding complete on first postpartum day (as shown by study of residual urine)	223	90.7	73	70.2	200	87.3
Voiding not complete on first postpartum day	23	9.3	31	29.8	29	12.7
Clinical urinary infections	0		1	0.96	4	1.7

* In 3 cases retention catheters were inserted and these cases are therefore not included.

† Table 3 shows the distribution of catheterizations among the groups.

TABLE 3.—*Distribution of Catheterizations*

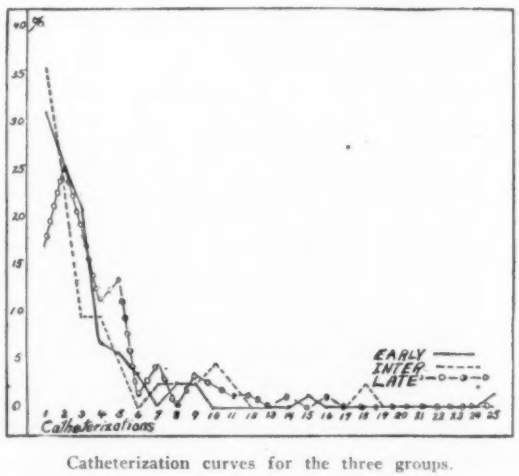
Number of Times Patients Were Catheterized	Early (87 Patients)		Intermediate (42 Patients)		Late (89 Patients)	
	No.	%	No.	%	No.	%
1	27	31	15	35.7	15	16.8
2	22	25.3	10	23.8	22	25.0
3	18	20.7	4	9.4	16	18.0
4	6	6.9	4	9.4	10	11.2
5	5	5.7	2	4.7	12	13.5
6	3	3.4	0	0	1	1.1
7	0	0	1	2.3	4	4.5
8	2	2.3	1	2.3	0	0
9	2	2.3	1	2.3	3	3.3
10	0	0	2	4.7	2	2.2
11	0	0	1	2.3	1	1.1
12	0	0	0	0	1	1.1
13	0	0	0	0	0	0
14	0	0	0	0	1	1.1
15	1	1.1	0	0	0	0
16	0	0	0	0	1	1.1
17	0	0	0	0	0	0
18	0	0	1	2.3	0	0
19	0	0	0	0	0	0
20	0	0	0	0	0	0
21	0	0	0	0	0	0
22	0	0	0	0	0	0
23	0	0	0	0	0	0
24	0	0	0	0	0	0
25	1	1.1	0	0	0	0

Of a total of 532 patients in our series delivered vaginally 481, or 90.4 per cent, were administered caudal block anesthesia, while 49 of a total of 50, or 98 per cent of patients delivered by cesarean section, were operated on under subarachnoid block.

In our entire series of 582 cases no changes in delivery or operative technic were made. All episiotomies were repaired routinely with number 0 and number 00 chromic catgut, without retention sutures. Abdominal incision closures were done routinely with number 0 and number 00 chromic catgut in continuous strands, with a few interrupted sutures placed in the fascia. No retention sutures were used, except when the incision was in the midline.

All patients requiring catheterization were catheterized at least one time for residual urine after voiding had begun, and, if this amount was above 50 cc., catheterization was repeated after each voiding until the amount of residual urine was 50 cc. or less. Patients who voided apparently normal amounts of urine without difficulty and at normal intervals were not catheterized at all. The estimations as to the amount of lochia or of the amount of postpartum bleeding were made from our observations during our rounds at the hospital, in addition to the reports of the obstetric nurses in attendance. Involution of the uterus was ascertained by a vaginal examination made on each patient at the time of discharge from the hospital.

All patients delivered vaginally remained in the hospital six to eight days, depending on the number of available beds, while the patients delivered by cesarean section remained eight to ten days. Only 12 patients, or 2 per cent of the entire series, stayed a few days beyond these limits, the majority of these because of minor complications. There were no serious complications in the entire series of 582 patients.



Catheterization curves for the three groups.

ANALYSIS OF STATISTICS

The study of bladder function, as shown in table 2, reveals that approximately 35 per cent of the early risers required catheterizations averaging three per patient, while approximately 40 per cent of the intermediate and 40 per cent of the late risers required catheterizations averaging four per patient. Table 3 shows that 77 per cent of the total catheterizations in the early group, 69 per cent in the intermediate group and 60 per cent in the late group occurred in the first three postpartum days. There were 5 cases of urinary infection, all of which occurred in the intermediate and late groups.

Analysis of the results in tables 2 and 3 suggests that there is no significant difference as regards bladder function in the three groups of patients.

A study of the chart shows the close similarity of the catheterization curves for the three groups.

19. At the Cedars of Lebanon Hospital the first postpartum day is defined as "the day following delivery."

The contention that early puerperal rising may cause excessive bleeding is disproved by the statistics, which actually show less frequent abnormal bleeding in the early group than in the late group, the intermediate group being approximately the same. Involution of the uterus was uniformly good in all groups.

The fear that early rising would cause a breakdown of episiotomy or abdominal wound incisions is likewise unfounded, only 1 instance of perineal skin separation occurring in the entire series, and this in a patient in the late rising group.

More efficient mobilization of the bowels is evident in table 4, over three times as many spontaneous bowel movements occurring in the early as in the late group.

The total number of complications in the entire series was very small, and there was no significant difference among the three groups. As there were no patients having emboli or thromboses in the entire series, the contention of many previous authors that late rising fosters these complications is not evident in our series. However, we are inclined to believe that study of a much larger series might possibly reveal a lower incidence of these complications in the early risers.

The claim advanced by some obstetricians that early rising may cause later prolapsus and retroversions is also disproved. Of early risers examined at one month and at six weeks, 28.7 per cent had either a midposition or retroversion of the uterus, while 34.6 per cent of the intermediate and 24.4 per cent of the late risers had the same positions—relatively similar totals. This indicates that early rising causes no significant alteration in the position of the uterus, at least within a period of four to six weeks following delivery. Prolapsus did not occur in any patient in the entire series.

TABLE 4.—*Miscellaneous Observations*

	Early, 247		Intermediate, 106		Late, 229	
	No.	%	No.	%	No.	%
Lochia:						
Normal	234	94.7	100	94.3	202	88.2
Moderate	11	4.5	6	5.7	24	10.4
Excessive	2	0.8	0	0	3	1.4
Perineal healing:						
Good	247	100.0	106	100.0	228	99.6
Fair	0	0	0	0	1*	0.4
Poor	0	0	0	0	0	0
Abdominal wound healing:						
Good	18	100.0	3	100.0	29	100.0
Fair	0	0	0	0	0	0
Poor	0	0	0	0	0	0
Involution of uterus:						
Good	244	98.8	102	96.2	228	99.6
Fair	3	1.2	4	3.8	1	0.4
Poor	0	0	0	0	0	0
Bowel function:						
Bowels moved spontaneously	65	26.3	17	16.0	18	7.9
First movement with enema on third day	182	73.7	89	84.0	201	92.1
Thrombosis	0	0	0	0	0	0
Embolism	0	0	0	0	0	0
All other complications:						
Mastitis	1	0.4	0	0	1	0.4
Stitch drainage	1	0.4	0	0	0	0
Endometritis	1	0.4	0	0	3	1.4
Postpartum hemorrhage	1	0.4	0	0	0	0
Cystitis	0	0	1	0.9	1	0.4
Pyelitis	0	0	0	0	3	1.4
Parotitis	0	0	0	0	1	0.4

* Skin separation only.

The nurses working in the obstetric department reported a decided reduction in the amount of nursing care required in patients rising early. They noted better morale and less discomfort in this group. Particularly favorable are their reports concerning cesarean sections, in which they have noted less abdominal distention and discomfort and a much more rapid convalescence.

No patient in either the early or the intermediate group offered any criticism to us concerning early rising. On the contrary, the majority exhibited enthusiasm, particularly because they did not have to use bed pans. The multiparas especially were impressed, stating that they felt better and stronger than they had after previous confinements, when they had spent seven to fourteen days entirely in bed.

TABLE 5.—*Postpartum Studies*

574 Studied Post Partum at Four and Six Weeks *

	Early, 244		Intermediate, 104		Late, 226	
	No.	%	No.	%	No.	%
Excessive bleeding during first month	8	3.3	2	1.9	9	4.0
Perineal support:						
Good	244	100.0	104	100.0	226	100.0
Fair	0	0	0	0	0	0
Poor	0	0	0	0	0	0
Position of uterus:						
Anterior	174	71.3	68	65.4	171	75.6
Middle	27	11.0	7	6.7	8	3.6
Retrograde	43	17.7	29	27.9	47	20.8
Complications during first month:						
Severe backaches	2	0.8	2	1.0	7	3.0
Psychotic episodes	3	1.2	0	0	4	1.8
Osteomalacia	0	0	0	0	1	0.4

* A total of 8 patients did not present themselves for postpartum study.

SUMMARY AND CONCLUSIONS

Our data indicate that delivered women can safely and advantageously get up early in the puerperium with no harmful results occurring.

Bowel function particularly is improved as a result of early rising in the puerperium.

Early puerperal rising reduces the amount of nursing care required.

The majority of patients managed in this way reported favorably on the method.

The statistics show no particular advantage to third or fourth day puerperal rising as compared to the earlier first and second day figures.

It is our opinion that early rising results in more rapid and comfortable convalescence, with less asthenia and less postoperative and postpartum depression.

It is our hope that this study will encourage further interest and continued observation on the question of early puerperal rising which may eventually clarify this still unsettled problem.

2007 Wilshire Boulevard.

Determination of Intoxication.—Blood reflects the concentration of alcohol in the brain more directly than does any other obtainable fluid. It has been a standard of reference in all scientific studies dealing with the amounts of alcohol in the body and the physiological effects from the alcohol. No particular difficulty is experienced in obtaining it, and there is no difficulty in the analytical determination of the alcohol. It is easily stored and easily transported. However, for blood, a physician is required to draw the sample and a trained technician to do the analysis. Over and above these requirements there are certain legal obstacles to the procurement of a blood sample. If it were not for these practical difficulties there is little doubt that all authorities would be in thorough accord on the use of blood as the material of choice in the determination of intoxication.—Greenberg, Leon A., in Alcohol, Science and Society, New Haven, Quarterly Journal of Studies on Alcohol, 1945.

ST. LOUIS ENCEPHALITIS

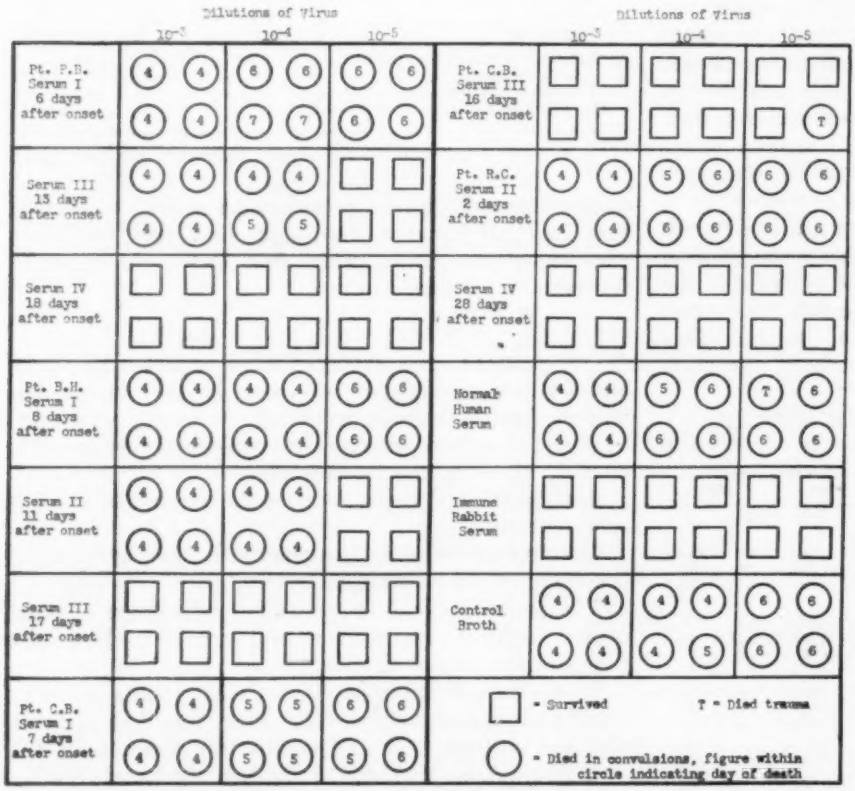
OCCURRENCE IN CHILDREN IN THE ST. LOUIS AREA DURING NONEPIDEMIC YEARS 1939-1944

RUSSELL J. BLATTNER, M.D.
AND
FLORENCE M. HEYS, PH.D.
ST. LOUIS

In the summer of 1933 an epidemic of acute encephalitis occurred in the St. Louis (Missouri) area. From August to November of that year there were 1,097 cases. A filtrable virus isolated from brain tissue of fatal cases was studied [1] extensively and was identified as the etiologic agent. In the summer of 1937 a second outbreak of acute encephalitis occurred in the St. Louis area. There were 750 cases, and a virus isolated from human brain tissue taken at autopsy was found to be immunologically identical with the infectious agent isolated in 1933. The occurrence of sporadic cases of this disease since 1937 indicates the existence of an endemic focus, a fact of considerable importance from an epidemiologic point of view.

Each year patients are admitted to the St. Louis Children's Hospital with symptoms and signs of acute encephalitis. Clinically it is difficult, if not impossible, to differentiate the types and establish the etiology with certainty. From the time of the 1937 epidemic, attempts have been made to ascertain by means of appropriate protection tests whether any of these cases of acute encephalitis was of the St. Louis type. Blood samples were obtained early in the course of the illness, at intervals throughout the hospital stay and at varying times after discharge from the hospital. The development during the clinical course of type specific antibodies to the St. Louis virus was studied by means of protection tests. In certain of the proved cases antibody studies were repeated on blood samples secured from one to three years after the acute attack.

Further efforts were made to differentiate the types of encephalitis by attempted isolation of an infectious agent directly from the spinal fluid. Accordingly, samples of freshly drawn spinal fluid were inoculated on the retracted chorioallantoic membrane of the developing hen's egg. If changes suggestive of viral growth were noted, usually in three to four days, membranes were triturated and were diluted with tryptose broth, and the supernatant fluid was inoculated intracerebrally into white Swiss mice. At the same time samples of spinal fluid were injected into white Swiss mice intracerebrally and intraperitoneally.


Dilutions of Virus
10⁻³
10⁻⁴
10⁻⁵
Pt. P.B. Serum I 6 days after onset
Serum III 13 days after onset
Serum IV 18 days after onset
Pt. B.H. Serum I 8 days after onset
Serum II 11 days after onset
Serum III 17 days after onset
Pt. C.B. Serum I 7 days after onset
Pt. C.B. Serum III 16 days after onset
Pt. R.C. Serum II 2 days after onset
Serum IV 28 days after onset
Normal Human Serum
Immune Rabbit Serum
Control Broth
= Survived
T = Died trauma
= Died in convulsions, figure within circle indicating day of death

Results of mouse protection test, showing increase in antibody titer to St. Louis encephalitis virus. Titration of virus used in this test showed the minimum lethal dose to be between 10^{-8} and 10^{-9}.

METHODS

Serum was withdrawn from blood samples under aseptic conditions and stored at a temperature of 4 C. St. Louis encephalitis stock virus (Hubbard strain) used for protection tests has been carried by intracerebral inoculation of white Swiss mice since its isolation at autopsy from human brain tissue. Repeated titration of the virus has shown that the minimal lethal dose for white Swiss mice remains between 10^{-8} and 10^{-9}.

MOUSE PROTECTION TESTS

Mouse protection tests, serum-virus neutralization, were used for the detection of antibody in the serum of a patient and for the demonstration of increasing titer of antibody during the clinical course of the disease.

Serum-virus mixtures were prepared by adding two parts of the patient's serum to one part of appropriate dilution of virus and incubating for two hours in a water bath at 36.5 C. Controls consisted of similar serum-virus mixtures prepared with normal human serum and serum from a rabbit immunized against the St. Louis virus, Hubbard strain. After two hours' incubation these mixtures were inoculated, 0.03 cc. intracerebrally, into adult white Swiss mice. The chart gives the results of a mouse protection test showing rising antibody titer during the course of the illness. Such titer of type specific antibody to the St. Louis virus, increasing during the clinical course, was accepted as evidence that the encephalitis was due to the St. Louis virus.

From the Department of Pediatrics, Washington University School of Medicine, and the St. Louis Children's Hospital.

1. Report on the St. Louis Outbreak of Encephalitis, Pub. Health Bull. 214, United States Treasury Department, Public Health Service, January 1935. Muckenfuss, R. S.; Armstrong, C., and McCordock, H. A.: Encephalitis: Studies on Experimental Transmission, Pub. Health Rep. **48**: 1341-1343 (Nov. 3) 1933. Webster, L. T., and Fite, G. L.: A Virus Encountered in the Study of Material from Cases of Encephalitis in the St. Louis and Kansas City Epidemics of 1933, Science **78**: 463-465 (Nov. 17) 1933.

TABLE 1.—*Clinical Manifestations in Cases of Acute Encephalitis Shown to Be of St. Louis Type*

Number	Year	Sex	Age, Yrs.	Symptomatology	Physical Examination	Spinal Fluid: Total Cells	Spinal Fluid: Lymphocytes, per Cent	Spinal Fluid: Pandy	Protection Test*: Mouse	Protection Test*: Egg
1	9/18/39 to 9/25/39	♂	9	History of tonsillitis, 1 week before admission Fever, 1 day Headache, 1 day	Temperature 39 C. Cervical rigidity	43	70	0	–	+
2	9/14/39 to 9/22/39	♂	9	Fever, 5 days Headache, 1 day Vomiting, 12 hours	Temperature 39 C. Cervical rigidity	100	100	0	+	+
3	5/14/40 to 5/20/40	♂	6	Headache, 4 days Fever, 3 days Drowsiness, 2 days Vomiting, 1 day Convulsions, mild, 18 hours	Temperature 39.5 C. Delirium, mild Slurred speech Cervical rigidity Kernig test positive Deep reflexes hyperactive Abdominal reflexes absent	200	92	+	+	+
4	10/30/42 to 11/ 9/42	♀	7	Headache, 5 days Vomiting, 5 days Fever, 5 days Stiff neck, 3 days Drowsiness, 2 days Dysphagia, 1 day Coma, 10 hours	Temperature 40 C. Comatose Cervical rigidity Opisthotonos Tremor Ocular nystagmus	65	100	+	+	+
5	8/26/42 to 9/10/42	♀	8	Headache and dizziness, 5 days Nausea, 5 days Fever, 5 days Malaise, 5 days Vomiting, 2 days Drowsiness, 2 days Stiff neck, 1 day	Temperature 40 C. Cervical rigidity Deep reflexes hyperactive Abdominal reflexes hyperactive	308	100	+	+	+
6	8/25/42 to 9/ 9/42	♀	8	Headache, 6 days Vomiting, 6 days Drowsiness, 4 days Leg pains for 2 days, 5 days before admission	Temperature 38 C. Cervical rigidity All deep reflexes hypoactive Abdominals absent Pronounced drowsiness	468	100	0	+	+
7	10/ 1/43 to 10/ 6/43	♂	5	Anorexia, 3 days Headache, 2 days Pain in neck, 1 day Drowsiness, 1 day Fever, 1 day Vomiting, 12 hours	Temperature 40 C. Slight cervical rigidity Drowsiness Positive Kernig test	750	48	+	+	+
8	8/16/43 to 8/27/43	♂	7	Anorexia, 5 days Malaise, 4 days Drowsiness, 4 days Headache, 3 days Fever, 3 days	Temperature 40 C. Cervical rigidity Pronounced drowsiness Positive Babinski, bilateral Kernig test positive	277	98	+	+	+
9	7/12/44 to 7/25/44	♀	12	Anorexia, 6 days Fever, 6 days Malaise, 6 days Headache, 6 days Nausea, 6 days Drowsiness, 5 days Disorientation, 3 days Convulsions, 1 day	Temperature 40.5 C. Lethargic Disoriented Convulsions Cervical rigidity Papilledema, bilateral Kernig test negative Babinski, equivocal Deep reflexes hypoactive	50	98	+	+	+
10	7/14/44 to 7/25/44	♀	6	Anorexia, 14 days Constipation, 9 days Intermittent fever, 8 days Muscular twitching, 5 days Vomiting, 4 days Leg pains, 2 days Headache, 2 days	Temperature 38.5 C. Pharyngitis, mild No neurologic findings except absent left upper abdominal reflex	90	60	–	+	+
11	7/24/44 to 7/30/44	♀	6	Anorexia, 6 days Headache, 6 days Otalgia, right, 6 days Drowsiness, 6 days Pain in legs, 6 days Pain in legs, severe, 5 days Stiff neck, 4 days Vomiting, 3 days Fever, 3 days	Temperature 38 C. Lethargic Cervical rigidity Facial paralysis, left Abdominal reflexes absent	50	80	–	+	+
12	9/ 7/44 to 9/13/44	♂	9	Headache, 2 days Fever, 2 days Stiff neck, 1 day Delirium, 12 hours	Temperature 38 C. Disorientation Cervical rigidity Positive Brudzinski, left Mild pharyngitis	186	18	–	+	+

* + = protection; 0 = no protection.
All patients were of the white race and were considered to have recovered at time of discharge.

TABLE 2.—*Distribution of Cases of Acute Encephalitis by Years and Months, 1938-1944*

	January	February	March	April	May	June	July	August	September	October	November	December	Totals
1938	..	..	..	1	..	1	4	2	2	..	..	..	10
1939	..	..	..	..	..	..	..	..	(2)	..	..	..	(2)
1940	..	..	2	..	(1)	2	..	7	2	..	..	..	13 (1)
1941	..	1	1	2	..	1	2	3	2	..	..	..	12
1942	2	3	..	..	1	1	1	(2)	1	(1)	2	..	11 (3)
1943	1	1	2	..	..	3	..	3 (1)	2	1 (1)	1	..	14 (2)
1944	..	..	..	..	4	1	2 (3)	..	2 (1)	3	2	2	16 (4)
Totals	3	5	5	3	5 (1)	9	9 (3)	15 (3)	11 (3)	4 (2)	5	2	76 (12)

Figures in parentheses indicate the number of cases shown to be of St. Louis type.

EGG PROTECTION TESTS

In each instance the results of the mouse protection tests were confirmed by means of egg protection tests. The egg protection test [2] furnishes a relatively simple and inexpensive method for detecting the presence of humoral antibodies and may be carried out by either of two procedures: (1) serum-virus neutralization or (2) protection by passive transfer. In the former, chorioallantoic inoculations consisted of 0.1 cc. of the serum-virus mixtures described. Such inoculated eggs were allowed to continue development for ninety-six hours following inoculation, after which the membranes were stained with trypan blue.[3] The degree of neutralization by a given serum sample is indicated by the reduction or absence of virus growth on the chorioallantois. Control membranes were inoculated with comparable mixtures of virus with normal human serum and with immune rabbit serum.

TABLE 3.—*Distribution of Cases of Acute Encephalitis According to Location and Year, 1939-1944, St. Louis Children's Hospital*

Cases Proved to Be St. Louis Type						
Years	1939-1940	1941	1942	1943	1944	Totals
St. Louis City	..	..	..	..	..	..
St. Louis County	3	..	2	1	1	7
Missouri	..	..	..	1	1	2
Illinois	..	..	1	..	2	3
Totals	3	..	3	2	4	12

Cases Not of St. Louis Type						
Years	1939-1940	1941	1942	1943	1944	Totals
St. Louis City	5	8	7	6	4	30
St. Louis County	3	2	..	3	5	13
Missouri	3	1	2	2	2	10
Illinois	1	2	2	1	5	11
Iowa	1	..	..	..	..	1
Louisiana	..	..	..	1	..	1
Totals	13	13	11	13	16	66

The passive transfer method consists in the passive protection of living cells by immune serum prior to inoculation with virus. Cells of the chorioallantois on contact with specific immune serum are rendered immune [4] to the corresponding virus, and on subsequent exposure virus growth is prevented. Serum was applied to the chorioallantois by means of an atomizer, 0.3 cc. of serum being distributed over the entire surface of the retracted membrane. Twenty-four hours after being sprayed with serum these membranes were inoculated with 0.1 cc. of virus suspension in serial dilution. Membranes sprayed with normal human serum and membranes sprayed with immune rabbit serum served as controls in each instance.

Serum-virus neutralization and passive transfer methods gave similar results, and both confirmed those obtained by mouse protection tests.

RESULTS

In 1938 Smith, Lennette and Blattner [5] reported 10 cases of acute encephalitis studied by mouse protection tests. None of these showed development of type specific antibody to the St. Louis virus.

From 1939 to 1944 inclusive 66 cases of acute encephalitis have been observed in St. Louis Children's Hospital and have been investigated for the development of type specific antibody to the St. Louis virus. Twelve of the 66 cases (18 per cent) showed increasing antibody titer to the St. Louis virus. The clinical findings of these 12 cases are summarized in table 1. Distribution of cases by years and months is presented in table 2. Ten cases reported by Smith, Lennette and Blattner in 1938 are included, bringing the total number of cases to 88.

It will be seen that approximately 74 per cent (65) of all the cases of acute encephalitis occurred in the period from May to October, inclusive; of the cases shown by increasing antibody titer to be St. Louis type, 100 per cent (all 12) occurred in May to October inclusive; 75 per cent of these 12 (9 cases) occurred within the three months period July, August and September.

In 2 of the proved cases of St. Louis type, blood samples obtained two and three years after acute illness showed high titer of antibody.

Table 3 gives the approximate geographic location of cases of acute encephalitis and their distribution by years. It will be noted that no case of the St. Louis type occurred in the city itself, whereas 30 cases which were shown not to be of the St. Louis type did occur in the city proper.

In no instance was the virus of St. Louis encephalitis isolated from spinal fluid either by egg or by mouse inoculation. In 1 case of acute encephalitis occurring in September 1944 the virus of lymphocytic choriomeningitis was recovered from the spinal fluid, and in another, occurring in January 1943, herpes simplex virus was isolated.

COMMENT

The occurrence of sporadic cases of St. Louis encephalitis in nonepidemic years in a district where two epidemics of this disease have occurred previously indicates the persistence of the infectious agent in the area. The fact that all these sporadic cases occurred during the warm months serves to reemphasize the seasonal distribution which was a striking feature of the disease during the epidemics of 1933 and 1937. It is of further interest that a significant number of the sporadic cases has occurred in districts where high per capita incidence was recorded during the epidemics.

While there has been considerable discussion relative to possible case to case transmission by droplet infection, it is significant that, despite numerous attempts, the virus has never been isolated from nasopharyngeal washings and that evidence for direct case to case transmission is lacking. As has been pointed out in a previous publication,[6] certain epidemiologic features of the disease suggest the possibility that blood sucking vectors may be important factors in its transmission.

2. Blattner, R. J., and Cooke, J. V.: The Use of the Egg Protection Test for the Recognition of St. Louis Encephalitis in Man, J. Infect. Dis. **70**: 226-230 (May-June) 1942. Blattner, R. J.; Heys, F. M., and Gollub, S. W.: Antibody Response to Cutaneous Inoculation with Vaccinial Virus in Human Subjects, Utilizing the Egg Technic: I. Serum-Virus Neutralization; II. Protection by Passive Transfer, J. Immunol. **46**: 207-215 (April) 1943.

3. Cooke, J. V., and Blattner, R. J.: Trypan Blue Vital Staining in Studies of Virus Lesions on Chorioallantoic Membranes, Am. J. Path. **18**: 163-167 (Jan.) 1942.

4. Blattner and Cooke.[2] Cooke and Blattner.[3]

5. Smith, M. G.; Lennette, E. H., and Blattner, R. J.: Etiologic Studies of Sporadic Cases of Encephalitis Occurring in the St. Louis Area in 1938, Am. J. Dis. Child. **59**: 509-514 (March) 1940.

6. Blattner, R. J., and Heys, F. M.: Blood Sucking Vectors of Encephalitis: Experimental Transmission of St. Louis Encephalitis (Hubbard Strain) to White Swiss Mice by the American Dog Tick Dermacentor Variabilis Say, J. Exper. Med. **79**: 439-454 (April) 1944.

Experimental work,[7] with an arthropod, the American dog tick Dermacentor variabilis, showed for the first time that the St. Louis virus can be transmitted to susceptible animals by the bite of such a vector and that the virus is passed by the infected female tick through her eggs and through all stages of subsequent development. Transovarian passage of the virus was followed into the third generation.

Hammon and his associates [8] demonstrated the presence of the St. Louis virus in mosquitoes (Culex tarsalis) collected in nature during epidemic periods and showed experimentally that the virus can be transmitted by the bite of culicine mosquitoes. However, persistence of the virus indefinitely in the mosquito has not been established.

More recently the presence of the St. Louis virus [9] has been demonstrated in the bodies of chicken mites (Dermanyssus gallinae) collected in nature from three separate localities in St. Louis County during nonepidemic years. These findings might serve to account for the existence of an endemic focus of the infectious agent of St. Louis encephalitis and the occurrence of sporadic cases of this disease in nonepidemic years. Details of epidemiology must await further field and experimental study.

SUMMARY

During the years 1939-1944 inclusive 66 cases of acute encephalitis were studied at the St. Louis Children's Hospital. Twelve were shown to be of the St. Louis type.

All 12 of these cases of St. Louis encephalitis occurred during the months May to October; 9 of the twelve occurred during July, August and September. The occurrence of sporadic cases in the St. Louis area indicates the existence of an endemic focus. Recent advances in the epidemiology of this disease suggest a possible explanation for the persistence of such an endemic focus.

St. Louis encephalitis virus was not recovered from spinal fluid. However, during the course of these investigations the virus of lymphocytic choriomeningitis was recovered from spinal fluid in 1 instance, herpes simplex in another instance.

500 South Kingshighway.

7. Blattner, R. J., and Heys, F. M.: Experimental Transmission of St. Louis Encephalitis to White Swiss Mice by Dermacentor Variabilis, Proc. Soc. Exper. Biol. & Med. **48**: 707-710 (Dec.) 1941; Blood Sucking Vectors of Encephalitis.[6]

8. Hammon, W. M.; Reeves, W. C.; Brookman, B., and Izumi, E. M.: Isolation of Viruses of Western Equine and St. Louis Encephalitis from Culex Tarsalis Mosquitoes, Science **94**: 328-330 (Oct. 3) 1941. Reeves, W. C.; Hammon, W. M., and Izumi, E. M.: Experimental Transmission of St. Louis Encephalitis Virus by Culex Pipiens Linnaeus, Proc. Soc. Exper. Biol. & Med. **50**: 125-128 (May) 1942. Hammon, W. M.; Reeves, W. C.; Brookman, B., and Izumi, E. M.: Mosquitoes and Encephalitis in the Yakima Valley, Washington: I. Arthropods Tested and Recovery of Western Equine and St. Louis Viruses from Culex Tarsalis Coquillett, J. Infect. Dis. **70**: 263-266 (May-June) 1942. Hammon, W. M.; Reeves, W. C., and Izumi, E. M.: Mosquitoes and Encephalitis in the Yakima Valley, Washington: II. Methods for Collecting Arthropods and for Isolating Western Equine and St. Louis Viruses, ibid. **70**: 267-272 (May-June) 1942. Bang, F. B., and Reeves, W. C.: Mosquitoes and Encephalitis in the Yakima Valley, Washington: III. Feeding Habits of Culex Tarsalis Coq., a Mosquito Host of the Viruses of Western Equine and St Louis Encephalitis, ibid. **70**: 273-274 (May-June) 1942. Hammon, W. M.; Reeves, W. C.; Brookman, B., and Gjullin, C. M.: Mosquitoes and Encephalitis in the Yakima Valley, Washington: V. Summary of Case Against Culex Tarsalis Coquillett as a Vector of the St. Louis and Western Equine Viruses, ibid. **70**: 278-283 (May-June) 1942. Hammon, W. M., and Reeves, W. C.: Laboratory Transmission of St. Louis Encephalitis Virus by Three Genera of Mosquitoes, J. Exper. Med. **78**: 241-253 (Oct.) 1943.

9. Smith, M. G.; Blattner, R. J., and Heys, F. M.: The Isolation of the St. Louis Encephalitis Virus from Chicken Mites (Dermanyssus Gallinae) in Nature, Science **100**: 362-363 (Oct. 20) 1944; Further Isolation of St. Louis Encephalitis Virus and Congenital Transfer of Virus in Chicken Mites (Dermanyssus Gallinae), Proc. Soc. Exper. Biol. & Med. **59**: 136-138 (June) 1945.

PRACTICAL ASPECTS OF PEPTIC ULCER MANAGEMENT UNDER SERVICE CONDITIONS

CAPTAIN JOHN RUSSELL TWISS
(MC), U.S.N.R.
AND
COMMANDER EUGENE V. PARSONNET
(MC), U.S.N.R.

Our purpose in this report is to present the results of an investigation of 82 consecutive cases of peptic ulcer among Naval and Marine Corps personnel, to review factors leading to disability and to formulate principles of diagnosis, management and disposition of military personnel having this condition. To the best of our knowledge no similar report relative to Naval personnel has been published. Because of the increasing prevalence of peptic ulcer among both civilians and military personnel in many of the principal countries at war,[1] a review of recent developments in this field seems justified.

The problems that beset the physician in civilian life in the diagnosis and management of peptic ulcer are well known. In military practice, however, additional factors tend to increase these difficulties. Any consideration of a patient in service must of necessity concern itself primarily with the disposition of the individual in respect to his ability to perform military duties. Particularly the Marine, by the nature of the service expected from him, must be one whose gastrointestinal tract can function well under stress and unusually arduous conditions of subsistence and diet.

GENERAL CONSIDERATIONS

The increasing importance of dyspepsia and particularly peptic ulcer as causes of disability in both civilian and military life has been stressed in many recent reports from England,[2] Canada,[3] Germany [4] and the United States.[5] In England the incidence proved high during heavy air raid periods and in forces having military reverses. In Germany, disability was prevalent in war workers and in military personnel before or immediately after going into front lines. In the United States the greatest increase in incidence of these disorders is noted in civilians [6] and in the Army. Hurst [7] concluded that "dyspepsia was the largest single disease in the British army, and the most important medical problem of this war."

Paper published under the auspices of the Section on Gastro-Enterology and Proctology.

This article has been released for publication by the Division of Publications of the Bureau of Medicine and Surgery of the U. S. Navy. The opinions and views set forth in this article are those of the writers and are not to be considered as reflecting the policies of the Navy Department.

1. Hurst, S. A.: Medical Diseases of War, Baltimore, William Wood & Co., 1940. Kirk.[13] Tidy.[12] Wilcox.[2] Hurst.[7] Miller.[4] Boles.[5] Hinton.[6]

2. Wilcox, P. H.: Gastric Disorders in the Services, Brit. M. J. **1**: 1008-1012 (June 22) 1940. Stewart, D. N., and Winser, D. M.: Incidence of Perforated Peptic Ulcer: Effect of Heavy Air Raids, Lancet **1**: 259 (Feb. 28) 1942. Gill, A. M.; Berridge, F. R., and Jones, R. A. Differential Diagnosis of Dyspepsia, ibid. **1**: 727 (June 20) 1942. Kirk.[13] Tidy.[12] Hurst (footnotes 1 and 7).

3. Feasby, W. R.: Peptic Ulcer in the Canadian Army (1940 to 1944), War Med. **6**: 300 (Nov.) 1944. Hincks, C. M.: War Work in Canada, Ment. Hyg. **25**: 10 (Jan.) 1941.

4. Miller, E.: The Neuroses in War, New York, Macmillan Company, 1940

5. Boles, R. S.: Observations on the Prevention and Management of Peptic Ulcer, J. A. M. A. **121**: 640 (Feb. 27) 1943. Hinton.[6] Walters and Butt.[10] Kantor.[9] Berk and Frediani.[11]

6. Hinton, J. W.: Incidence of Peptic Ulcer and Its Complications, Am. J. Surg. **20**: 102 (April) 1933.

7. Hurst, A.: Digestive Disorders in Soldiers, Am. J. Digest. Dis. **8**: 321 (Sept.) 1941.

Peptic ulcer constitutes an important problem in the armed forces because experience in all military services has shown that in general patients with this condition are unfit for service and that a large proportion of these patients had the condition prior to enlistment.[8] The reported incidence of peptic ulcer among all gastrointestinal diseases is variable. In England it is reported as from 35 to 58 per cent,[9] in Canadian soldiers invalided home 21 per cent,[10] in United States Army hospitals from 7.2 per cent to 43 per cent,[11] in a South Pacific combat zone hospital 19 per cent. In England it has been estimated that 90 per cent of the patients had the condition before enlistment,[12] in the United States the reported incidence is from 50 to 93 per cent.[13] Duodenal ulcer is far more prevalent than gastric ulcer. The ratio of gastric to duodenal ulcers in the British army is reported as 1:3.6;[12] in the Army hospitals of the United States it varies from 1:8 to 1:49.[14] In Naval personnel the proportion was 1:9.[15]

ETIOLOGIC FACTORS

The cause of peptic ulcer is uncertain notwithstanding extensive physiologic, psychiatric and experimental studies.[16] Important contributing factors are conceded to be the excessive secretion of gastric hydrochloric acid and pepsin as well as gastric hypersecretion and hypermotility. All these conditions have resulted from excessive and at times prolonged stimulation of the vagus nerve, either from neurogenic or from psychogenic causes.[17] The same vagus stimulation has been found to produce circulatory changes in the gastric mucosa consisting of anoxia, increased capillary permeability, tissue edema and decreased circulating blood volume.[18] With the physiologic changes described, peptic ulcers have been observed and produced repeatedly.

Psychosomatic medicine has made a great contribution to the practical understanding and management of peptic ulcer by demonstrating the important part that emotional disturbances can play in the formation and recurrence of peptic ulcer.[19] That the physiologic disturbances described which are associated with peptic ulcer can result from emotional disturbances has been demonstrated by Wolf and Wolff[18] in a laboratory assistant having a permanent gastrostomy opening. Repeated observations in this individual indicated that feelings of hostility, anger or resentment resulted in vigorous contractions of the stomach, a sharp increase in gastric acidity and secretion, and a turgid hyperemic gastric mucosa typical of hypertrophic gastritis. This state was found to be associated with symptoms of pain and heartburn. The turgid mucosa furthermore proved to be abnormally susceptible to injury, ulceration and hemorrhage and to be hypersensitive to direct stimulation.

Palmer[20] has contributed a valuable psychosomatic analysis of the factors in the formation of peptic ulcer. He states that heredity and dietary factors, as well as psychosomatic relationships, must be taken into consideration in the study and management of patients with peptic ulcer. The hereditary contribution, frequently overlooked, is that the ulcer patient has a physique which limits his behavior to a certain percentage of his aspirations, which are usually excessively high. The intolerance of these patients to certain types of food is well known. The psychosomatic relationships are primarily concerned with the higher centers and the stomach, which acts in two capacities. One is concerned with its reflex response to foods. The other function is as "an effector unit in the total behavior pattern," in which the stomach responds to psychic stimuli by muscle contraction and gland secretion.

A practical application of the principles described by Palmer is furnished in a psychiatric and physiologic study by Mittelman, Wolff and Scharf[21] in a series of 30 patients with peptic ulcer. All these patients appeared to be successful and without nervous disorders or life problems. Actually, all proved to have backgrounds of anxiety, insecurity, resentment and hostility. Under usual conditions of living they were relatively comfortable and effective. However, under unusual conditions of stress or disturbing situations produced experimentally, feelings of insecurity and anxiety occurred. These emotional disturbances were almost always associated with an increase in gastric acidity and pepsin secretion. Gastric peristaltic action was greatly increased and frequently pain and sometimes hemorrhage occurred. These investigators conclude that the emotional disturbances and functional changes in the stomach are not cause and effect but "coincident aspects of biologic behavior in reaction to life situations, behavior in some cases being compatible with health, in others resulting in disease."

The importance of these investigations in the diagnosis and treatment of service personnel is evident. Assuming that the so-called "ulcer diathesis" is the unstable type of individual described, the effect under adverse conditions of new situations, new diets (especially K rations) and environmental changes of a military nature may well be reflected in the disability of the

8. Graham, J. G., and Kerr, J. D. O.: Digestive Disorders in the Forces, Brit. M. J. **1**: 473 (March 29) 1941. Payne, R. T., and Newman, C.: Interim Report on Dyspepsia in the Army, ibid. **2**: 819 (Dec. 14) 1940. Tidy.[12]

9. Kantor, J. L.: Digestive Diseases and Military Service, J. A. M. A. **120**: 254 (Sept. 26) 1942.

10. Urquhart, R. W. I.; Singleton, A. C., and Feasby, W. R.: The Peptic Ulcer Problem, Canad. M. A. J. **45**: 391 (Nov.) 1941.

11. Chamberlin, D. T., and Wallace, W. C.: Perforated Peptic Ulcer in an Army General Hospital, Mil. Surgeon **92**: 193 (Feb.) 1943. Palmer, W. L.: The Stomach and Military Service, J. A. M. A. **119**: 1155 (Aug. 8) 1942. Berk, J. E., and Frediani, A. W.: The Peptic Ulcer Problem in the Army, Gastroenterology **3**: 435 (Dec.) 1944, and the articles listed in footnote 13.

12. Tidy, Henry: Peptic Ulcer and Dyspepsia in the Army, Brit. M. J. **2**: 473 (Oct. 16) 1943.

13. Kirk, R. C.: Peptic Ulcer at Fort Sill, Am. J. Digest. Dis. **10**: 411 (Nov.) 1943. Chamberlin, D. T.: Peptic Ulcer and Irritable Colon in the Army, ibid. **9**: 350 (Aug.) 1942. Flood, C. A.: Peptic Ulcer at Fort Meade, War Med. **3**: 160 (Feb.) 1943. Rush, A.: Gastrointestinal Disturbances in the Combat Area: I. Preliminary Observations on Peptic Ulcer, J. A. M. A. **123**: 389 (Oct. 16) 1943. Schildkraut, H.: Management of the Dyspeptic Soldier in a Staging Area, War Med. **6**: 151 (Sept.) 1944. Zetzel.[14]

14. Zetzel, L.: Experiences with Peptic Ulcer in an Army Station Hospital, Gastroenterology **3**: 472 (Dec.) 1944.

15. Walters, W., and Butt, H. R.: Management of Ulcers Among Naval Personnel, Ann. Surg. **118**: 489 (Oct.) 1943.

16. Dragstedt, L. R.: Pathogenesis of Gastroduodenal Ulcer, Arch. Surg. **44**: 438 (March) 1942. Berk, J. E.; Thomas, J. E., and Rehfuss, M. E.: The Acid Factor in Duodenal Ulcer, as Evaluated by the Acidity and Neutralizing Ability in the Duodenal Bulb, Am. J. Digest. Dis. **9**: 371 (Nov.) 1942. Schiffrin, M. J., and Ivy, A. C.: Physiology of Gastric Secretion, Particularly as Related to the Ulcer Problem, Arch. Surg. **44**: 399 (March) 1942. Cannon, W. B.: The Influence of Emotional States on the Function of the Alimentary Canal, Am.J. M. Sc. **137**: 480, 1909. Winkelstein (footnotes 22 and 17).

17. Winkelstein, A.: Some Observations on the Relationship of the Vagus Nerve to Peptic Ulcer, J. Mount Sinai Hosp. **9**: 859 (Nov.-Dec.) 1942. Boles, R. S., and Riggs, H. E.: Neurogenic Factors in the Production of Acute Gastric Ulcer, J. A. M. A. **115**: 1771 (Nov. 23) 1940.

18. Wolf, S., and Wolff, H. G.: Human Gastric Function: An Experimental Study of a Man and His Stomach, London, Oxford University Press, 1943. Boles and Riggs.[17] Mittelmann, Wolff and Scharf.[21]

19. Weiss, E., and English, O. S.: Psychosomatic Medicine, Philadelphia, W. B. Saunders Company, 1943. Kennedy, F.: Functional Nervous Disorders Associated with Warfare, New York State J. Med. **42**: 425 (March 1) 1942.

20. Palmer, D.: Psychosomatic Considerations in Peptic Ulcer, Ohio State M. J. **35**: 717 (July) 1939.

21. Mittelman, Bela.; Wolff, H. G., and Scharf, Margaret D.: Emotions and Gastroduodenal Function: Experimental Studies on Patients with Gastritis, Duodenitis and Peptic Ulcer, Psychosom. Med. **4**: 5 (Jan.) 1942.

patient. That the civilian can frequently accommodate himself to circumstances of his own making indefinitely but may quickly break down in service is shown by Tidy's comparative studies of civilian and military personnel with peptic ulcer in England.[12]

CLASSIFICATION

Our study is based on a consecutive series of patients with peptic ulcer admitted to the gastroenterologic ward of a large Naval hospital from Nov. 1, 1944 to March 28, 1945. During this time 5,379 patients were admitted to the hospital, of whom 238, or 4 per cent, had disease of the gastrointestinal tract. Of the latter number 82 have been proved cases of duodenal or stomach ulcers, or 35 per cent of all gastrointestinal diseases. Of these, 72 were duodenal and 10 gastric, the ratio of gastric to duodenal ulcers being 1:7. We have further subdivided these groups into those which were service incurred and those in which there was definite evidence that the illness had its onset prior to entry into service.

Duodenal Ulcer.—Service Incurred: Forty of our total of 72 duodenal ulcers, or 55 per cent, were incurred since the patients' enlistment (table 1). An analysis of the statistics reveals the average age in this group to be 28 years and the average duration of symptoms one year and ten months. The average time elapsing between enlistment and the onset of symptoms was two years. Thirty-nine of the 40 patients gave a history of pain and 36 had a typical pain food-relief-syndrome. Twenty-six had nausea or vomiting or both. Hyperacidity was present in only 14, and 15 either gave a history of hematemesis or had at some time had positive laboratory findings of occult blood in the stool. Positive x-ray evidence of activity or residual deformity was found in 39 cases.

Not Service Incurred: Of the 72 cases of duodenal ulcer, there were 32 cases, or 45 per cent, in which there was definite evidence that the condition antedated service (table 1). The average age of this group is 28 and the average duration of symptoms six and one-half years. The average time between enlistment and recurrence of symptoms was six months. Thirty-one of the 32 patients had epigastric pain and 28 gave a history of typical food relationship. Nausea or vomiting has been present in 22 cases. Hyperacidity was found in 14 of the 32 cases and evidence of bleeding in 16. The x-ray diagnosis was positive in all of this group.

Stomach Ulcer.—Service Incurred: Of the 10 stomach ulcers, 6 were incurred in service. The youngest patient was 20 and the oldest 28, and the average duration of symptoms was thirteen months. Five of this group gave a history of pain, 4 of a typical food relationship and 4 of nausea and vomiting. Four had hyperacidity, whereas 3 had evidence of bleeding. Two of these patients had negative x-ray findings; the diagnosis in 1 case was made on the finding of a large prepyloric ulcer during the course of a cholecystectomy for cholelithiasis, and in the second case the diagnosis was based on a classic history and two episodes of severe hemorrhage, for which no other demonstrable cause was found.

Not Service Incurred: In the remaining 4 cases ulcer existed prior to enlistment. The youngest was 19 and the oldest 26, and the average duration of symptoms was five years. All 4 patients had pain, 3 food relationship and 2 nausea or vomiting. Only 1 had hyperacidity, and 2 had evidence of bleeding. All had positive x-ray findings.

The number of stomach ulcers is too small from which to draw any definite conclusions, but of the patients with histories of ulcer antedating service all had shorter intervals of quiescence between enlistment and recurrence than the interval between enlistment and onset of symptoms in the service incurred cases.

DIAGNOSTIC CRITERIA

The important diagnostic criteria are a history of pain, the classic pain-food-relief syndrome and associated nausea or vomiting. Chronicity and periodicity are features that can be elicited frequently. Physical examination is rarely of significance, although many of these patients have a spastic, tender sigmoid. Hyperacidity and hypersecretion are always suggestive, and any evidences of bleeding demands the most careful consideration. Finally, the most conclusive finding is made by x-ray studies.

In the entire series only 3 gave no history of pain, and 2 of these were cases in which the presenting symptom was either hematemesis or tarry stools. Seventy-

TABLE 1.—*An Analysis of the Histories and Findings in the Series of 82 Patients Having Peptic Ulcer*

	Number of Cases	Average Age	Average Duration of Symptoms	Number of Patients Having					
				Pain	Food Relation	Nausea or Vomiting	Hyperacidity	Evidence of Bleeding	Positive X-ray Findings
Duodenal Ulcer									
Service incurred	40	28 (20-46)	22 mo. (6 wk. to 8 yr.)	39	36	26	14	15	39
Not service incurred	32	28 (18-49)	6½ yr. (2 yr. to 17 yr.)	31	28	22	14	16	32
Totals	72			70	64	48	28	31	71
Stomach Ulcer									
Service incurred	6	25 (20-28)	13 mo. (1 mo. to 2½ yr.)	5	4	4	4	3	4
Not service incurred	4	22 (19-26)	5 yr. (2½ yr. to 7 yr.)	4	3	2	1	2	4
Totals	10			9	7	6	5	5	8

one of the 82 patients were relieved of pain by the ingestion of bland foods. These predominant symptoms, pain and food relief, and particularly bleeding, frequently justify a diagnosis of ulcer, notwithstanding negative x-rays. Corroborative evidence is added by the finding of hyperacidity and (or) hypersecretion, but our observation is that under service conditions such findings are relatively infrequent. We consider gastric contents with over 30 degrees free hydrochloric acid on a fasting stomach or over 40 degrees free hydrochloric acid after a bread test meal to be definitely hyperacid. Hyperacidity was present in 28 of the 72 cases of duodenal ulcer and in 5 of the 10 cases of gastric ulcer. Hypersecretion was present in very few instances.

It will be noted that in only 3 of our patients was there absence of x-ray evidence of ulcer. Such evidence is the finding of a persistent niche with crater, or constant deformity. Frequently in cases presenting vague, atypical histories and with no laboratory findings indicative of ulcer the diagnosis has been based solely on the findings of the roentgenologist. Therefore we feel that despite our best efforts there unquestionably have been some patients whom we have returned to duty with existing but unproved organic lesions.

TREATMENT

The treatment shall be described in slight detail, since we believe that the general pattern is followed with little variation in the majority of institutions. The general principles have been described by Winkelstein [22] and Aaron.[23]

All patients admitted with an established diagnosis, or in whom the existence of ulcer is strongly suspected, are put on complete bed rest with only bathroom privileges, a diet of milk and cream every two hours, medication consisting of Amphojel or other antacid medication, belladonna, mild sedation and supplementary vitamins. Bed rest is continued for a two week period. The diet is increased each week until at the end of three or four weeks patients are taking a full modified type of Sippy diet. Medication and smoking restriction are continued throughout the course of treatment.

The length of time for treatment and modifications of the routine vary according to the individual response. The degree of improvement is estimated on the basis of the amelioration of subjective symptoms and the findings of an x-ray study performed six weeks following the original x-ray. Whereas in some cases it may be necessary to prolong the period of definite dietary restriction or bed rest, nevertheless the majority of patients show evidence of rapid improvement and at the end of two or three weeks complain only of vague and occasional epigastric distress. Outside "liberty" with its temptations to dietary and other indiscretions is not granted until the patient is free from symptoms. Medical treatment is continued until the patient is able to tolerate a full Sippy diet without medication, a regular diet with certain restrictions as related to foods which the individual finds he does not tolerate well, or until it is established that there exist certain indications for operative intervention.

TABLE 2.—*Summary of the Disposition of 82 Cases of Peptic Ulcer*

	Number	Full Duty	Limited Duty	Discharged
Duodenal Ulcer				
Service incurred:				
Marines	32	0	5	27
Naval	8	0	5	3
Totals	40	0	10	30
Not service incurred:				
Marines	23	0	0	23
Naval	9	0	2	7
Totals	32	0	2	30
Stomach Ulcer				
Service incurred	6	0	4	2
Not service incurred	4	0	0	4
Totals	10	0	4	6

As concerns operative treatment, we are in accord with the writings of Lahey.[24] At the writing of this report one of us has performed subtotal gastrectomies on 4 patients in this series. Three were duodenal ulcers, in 2 of which there was almost a complete lack of response to medical treatment after a month of rigid routine, the third case having been one in which two severe episodes of intestinal bleeding had occurred and in which an interval operation was performed. In the fourth case a large prepyloric crater was found during the course of a cholecystectomy, and a resection was performed. The type of operation is modeled after that described by Hofmeister, and we prefer the antecolic, long loop anastomosis. All 4 patients made uneventful operative recoveries, but it is too early at this date to estimate the ultimate results. We should like to substantiate the findings of other surgeons who have found that early mobility greatly aids in the immediate postoperative period. Our patients are permitted and encouraged to move about after the effects of the anesthesia have subsided. They are given bathroom privileges at once and by the third postoperative day are able to walk about freely with but little discomfort.

It is important at this time to mention a factor in the treatment of ulcer patients that is of the greatest value, namely the control of exciting emotional factors. We have found the most striking evidence of this in patients in whom for one reason or another there has been a failure to make a diagnosis. Such patients are sometimes told that they have "nervous stomachs," are "unstable" or just have "nothing the matter with them." The improvement invariably is most noticeable and pronounced, once the patient discovers that he has some tangible disease which is expected to respond to treatment.

The individuals coming under our observation have varied in age between 18 and 46. They have been white and black, tall and short, fat and thin, nervous and phlegmatic. There is no common physical habitus, but the one common denominator is an unstable or maladjusted psychogenic and physiologic background. Marital difficulties, money matters, uncertainty as to ultimate disposition are only a few of the great number of problems that make so difficult the treatment of such patients. We have made as great an attempt as our ability and time permit to try to understand the individual and to be of assistance to him in allaying the exciting psychic factors.

RESULTS OF TREATMENT AND DISPOSITION

The results of treatment are interpreted primarily by clinical response and x-ray findings. We have had occasion to make follow-up x-ray studies in 46 instances. These studies, made at the end of six weeks of treatment, revealed no evidence of any organic lesion in 20 cases, a lessening in the size of the crater in 10, residual deformity but no crater in 8 and demonstrated no change in 8. It has been observed in a number of our cases that the clinical improvement is not proportionate to the findings of repeated x-ray examination; some patients who are symptom free still having positive x-ray findings, and others with a negative x-ray interpretation still complain of clearcut subjective symptoms. We classify our clinical results in accordance with the patient's ability to tolerate various levels of diet.

Of 40 patients with service incurred duodenal ulcer under treatment for six weeks, 26 were able to tolerate a full Sippy diet without medication and without discomfort, 3 were partaking of a regular hospital diet without complaints, 9 have shown slight improvement and 2 showed no improvement. Thirty-two of these 40 patients were Marine personnel. Of those, 26 have been discharged from service and 3 were returned to "limited duty." (This term means duty limited to the continent of the United States.) There were in this

22. Winkelstein, A.: The Modern Treatment of Peptic Ulcer, Bull. New York Acad. Med. **20**: 87 (Feb.) 1944.
23. Aaron, A. H.: Inflammatory Lesions of the Upper Gastrointestinal Tract, J. A. M. A. **127**: 1027 (April 21) 1945.
24. Lahey, F. H.: Inflammatory Lesions of the Stomach and Duodenum, J. A. M. A. **127**: 1030 (April 21) 1945.

group 3 with subtotal resections, 2 of whom were returned to limited duty, the other having been discharged from service. Naval personnel in this classification numbered 8. Three have been discharged from service, 5 were returned to "limited duty," 1 having had a gastroenterostomy performed. In the latter instance a partial gastrectomy was deemed inadvisable because of the extensive inflammatory reaction surrounding the lesion (table 2).

Of 32 patients with duodenal ulcer incurred prior to enlistment, 11 showed slight improvement and the remainder were able to tolerate a full Sippy diet. Practically none, however, were able to tolerate a regular hospital diet, which is the one primary prerequisite for return to duty. Of these 32 patients 23 were Marines, all of whom were discharged from service. Of the 9 Naval personnel, 7 were discharged from service and 2 were returned to limited duty.

In 6 of the 10 patients with stomach ulcer the ulcer was service incurred. Of these, 2 had been operated on for perforation prior to their admission to this hospital and were discharged from service able to tolerate a full Sippy diet. One patient was operated on at this hospital for cholelithiasis, and during the operation a large prepyloric crater was found and a subtotal resection was performed. This patient has returned to limited duty. The remaining 2 patients were discharged from service, 1 as slightly and the second as much improved.

In the remaining 4 patients with stomach ulcer the ulcer was not service incurred. All 4 have been discharged from service having shown considerable improvement under medical treatment, although in 2 of these cases, in spite of normal gastric analyses and gastrointestinal x-ray studies, there were still present mild subjective complaints on a moderately restricted diet.

A further breakdown of our statistics shows that there have been 63 Marines and 19 Naval personnel in this series. Of the 63 Marines, 57 have been released from service and 6 retained. Of the 19 Naval personnel, 12 have been discharged and 7 retained. These figures reflect quite accurately our estimates as to the different character of duty required by these two branches of service.

It also will be noted that in the 82 patients with peptic ulcer the ulcers of 46 were service incurred. Thirty-five of these have been released from service and 11 have returned to limited duty. Among the 36 patients with ulcer not service incurred, only 2 have returned to duty whereas the remainder have been discharged. Thus we see the somewhat less favorable response to treatment in those cases in which the symptoms are of longer duration.

There remains to discuss the contributory effects of service conditions to the incidence of ulcer. Among the patients with duodenal ulcer the average length of time between enlistment and onset of symptoms in patients having had duodenal ulcer prior to enlistment was approximately six months, whereas in those having ulcers incurred in line of duty the average period between enlistment and disability was two years. Few of our patients had reached combat areas and rarely was the onset of symptoms related to such conditions. However, field conditions and particularly field "chow" frequently precipitated digestive symptoms. The conclusions which seem justified by these facts are that the psychogenic constitutional states of these individuals, together with problems of military service and the attendant physiologic disturbances, are the essential factors in the production of their ulcers. We further feel justified in stating that the attendant emotional and physical stress placed on many of these already unstable and (or) maladjusted individuals has caused an earlier incidence of symptoms than might have been the case during the uninterrupted course of an adjusted civil life.

Finally, as a result of our experience we are convinced that any individual with a history of ulcer should not be admitted to service. The short period of time elapsing in the average case between enlistment and disability precludes any great degree of usefulness on the part of the patient and is certain to cause a great many sick days and to render return to active civilian work complicated and uncertain. Furthermore, the disposition of patients with service incurred lesions can be decided on in a shorter time than has been customary heretofore. We recommend early separation from service in these cases also, especially if the individual shows any signs of psychosomatic disturbance such as emotional instability, unless his military duties are of a very specialized, noncombative character.

SUMMARY AND CONCLUSIONS

1. The ulcers in this series of 82 Marine and Naval personnel patients were incurred before enlistment in 55 per cent of the cases, in line of duty in 45 per cent. Disability occurred in an average time of two years in patients with duodenal ulcer not having the condition previous to enlistment. Those having a history of previous ulcer were disabled within six months. Few reached any combat areas.

2. The most reliable diagnostic criteria are considered a history of pain, particularly related to meals, evidence of hemorrhage and positive x-ray findings of ulcer.

3. Treatment has consisted in the use of a progressive modified Sippy type of diet with initial feedings every two hours, antacids, belladonna and vitamin supplements.

4. All patients are treated for a minimum period of six weeks with an initial period of two weeks' bed rest. Smoking is prohibited. No liberty is granted until the patient is symptom free and able to tolerate a regular diet.

5. No patient is considered a candidate for limited duty unless he can tolerate a regular diet without medication and has no evidence of an unstable emotional background.

6. Patients with proved peptic ulcer are usually discharged from the service. Limited duty within the continental limits of the United States has been recommended in a limited number of cases if the individual is particularly desirous of returning to duty, is unusually qualified from the point of view of training or experience, and there is a reasonable likelihood that the type of duty to which he returns is not too arduous and will permit of a certain amount of dietary discretion.

7. No patient with a history or findings of peptic ulcer, in our opinion, should be admitted to the armed forces. Early separation from service is recommended in all cases of peptic ulcer.

ABSTRACT OF DISCUSSION

DR. SARA M. JORDAN, Boston: Of considerable significance for postwar civilian life is the difference noted in average time between enlistment and onset of symptoms in the service incurred group (two years) and between enlistment and recurrence of symptoms in the group of ulcers not service incurred (six months). These data suggest that in persons who have an ulcer diathesis, an actual occurrence of ulcer increases the susceptibility. In my clinical experience I have been impressed with this fact and for this reason have stressed the importance of prophylaxis of this disease in ulcer prospectives. A serious effort should be made to detect those individuals who have symptoms of pylorospasm or hyperchlorhydria and x-ray evidence of pylorospasm and duodenal spasm, and, having detected them, we should make every effort to protect them against actual occurrence of ulcer with good hygiene, a reasonably careful diet and the omission of smoking. It is also important to treat ulcer intensively at its first occurrence and in this treatment to include adequate instruction concerning subsequent prophylaxis. Those of us who see ulcer complications in their most disastrous forms are constantly impressed with the fact that early intensive care could have prevented them. There is undoubtedly much less overtreatment than undertreatment of early ulcer. As Drs. Twiss and Parsonnet have indicated, the diagnosis of ulcer must often be made without the actual demonstration of a crater. Irritability of the duodenum with no actual ulcer defect may be the only radiologic finding, but, if this is associated with the subjective findings of ulcer, treatment should be instituted. Very often later x-ray examination shows a constriction in the second portion of the duodenum or an actual retraction deformity of the cap. The disappearance of irritability is, in my experience, the best x-ray guide to improvement and healing.

COLONEL JOHN L. KANTOR, M. C., A. U. S.: This report shows that Navy experience with peptic ulcer coincides closely with that of the Army. It is unfortunate that so many ulcer subjects have been inducted into the military services. Forty-five per cent of the authors' cases fall into this category, and this seems pretty much the general experience. Can something more than the present screening at induction stations be done to improve the situation? About ten years ago I recommended (*Mil. Surgeon* **74**:113 [March] 1934) that inductees presenting a history of ulcer be handled as follows: A certain number might be disposed of at draft or examining boards, where it is merely a question of checking up on readily accessible data such as previous operations or diagnoses by reliable physicians or hospitals. All other ulcer suspects or ulcer claimants should be immediately hospitalized in army or navy general hospitals, where sections of gastroenterology would already be set up and in operation. It would then be the duty of the gastroenterologists to make appropriate studies and to decide between immediate rejection or acceptance for full or for limited service. By not allowing such inductees to perform any military or naval duty before hospitalization and by promptly discharging all proved ulcer cases, the service aggravation of preexisting ulcers would be greatly reduced if not completely eliminated, thus saving the government large sums that would otherwise be paid out to meet disability claims. As to the disposition of ulcer patients, I am in general agreement with the authors. Non-service incurred cases should be discharged unless the subject is so valuable to the service that he can be given special opportunities to follow a proper diet and work in a favorable environment. Service incurred cases should be strictly individualized. Some will have to be discharged, to be sure; yet it should be noted that the arbitrary dismissal of a professional soldier or sailor, especially in the case of an officer, is as much of a psychic shock to him as is the retention in service of an unhappy civilian. In short, every effort should be made to retain trained veterans whose services are needed and who desire to continue on active duty.

CAPTAIN A. M. SNELL (MC), U.S.N.R.: The statistics on this subject, as presented by the authors, appear to be in close agreement with the general impressions which I have gathered from eighteen months' observation of patients in the gastrointestinal service of a West Coast naval hospital. Roughly, two thirds of our patients had ulcer symptoms prior to enlistment, a fact which is not always given in the health record and which not infrequently requires investigation and verification by the Red Cross authorities near the patient's home. A large percentage of our patients have had foreign duty, and many of them have been in actual combat. There is no doubt that many of them would have carried on successfully in civilian life had not the psychogenic factors mentioned by the authors intervened. Living conditions on ships and shore stations in combat areas, the arduous duties required of many of our patients and their anxiety about their own situation and that of their families are obviously responsible for the reactivation of many quiescent ulcers. Return to the United States, a brief period of leave and a more normal mode of life are productive of complete symptomatic relief in many patients, and neither prolonged medical treatment nor surgery is often required. The authors mention "anxiety, insecurity, resentment and hostility" in their discussion of psychosomatic factors; the importance of these factors in men in combat areas can hardly be overestimated. In a not inconsiderable number the existence of an ulcer becomes, either consciously or subconsciously, a means of escape from a hazardous and unpleasant existence. I have been impressed at this hospital with three other matters of clinical importance: (1) the high percentage of patients who relate bizarre or atypical histories, although roentgen evidence of ulcer is indisputable; (2) the relatively small number of patients who have neither pronounced hyperchlorhydria nor hypersecretion, and (3) the relative rarity of complications. Free perforations or inflammatory penetrating lesions are rarely seen; hemorrhage of a major degree is not often a problem; and obstruction, except that of a transient nature incidental to pylorospasm, is almost unknown. I agree with the authors that the man with a peptic ulcer rarely has a useful place in military service. A few devoted individuals carry on in spite of an ulcer, and one occasionally encounters officers or petty officers who have performed long and useful duty both before and after necessary gastroduodenal surgery. To do so obviously requires a temperament not ordinarily encountered in an ulcer-bearing individual.

DR. A. H. AARON, Buffalo: One is impressed by the evidence indicating the etiologic importance of psychiatric trauma in peptic ulcer. There is definite agreement today that this results in alterations in secretion, motility and tone of the gastrointestinal tract conducive to peptic ulcer formation. It is interesting to note that 32 of the authors' patients evidently had peptic ulcer previous to their entrance into the armed forces. It seems difficult to explain how they were accepted, but having worked for a period on an induction team I can readily testify to the difficulty of obtaining an adequate history that would suggest the presence of peptic ulcer. Even after having secured such a history, one frequently finds the x-ray findings negative, and in view of this it was impossible on the suggestive history alone to exclude the individuals from military service. The symptoms of nausea and vomiting have not impressed me as of diagnostic significance in uncomplicated peptic ulcer such as we see in young adults with an average history of slightly more than a year's duration. These symptoms are more often an indication of the complicating factor of pyloric obstruction due to edema, spasm or scar tissue. I wonder if the incidence of these symptoms in their group of cases bears any relation to what has appeared in the literature as manifestations of acute psychiatric upsets occurring during the course of the war effort. I agree that medical treatment must aim toward as near as possible control of the acid peptic secretion and protection against psychiatric shock. From the surgical point of view I think the group of irreplaceable individuals on whom Walters and Butt did a resection relatively early in order to shorten the period of treatment is a very significant event and one which we may have to apply occasionally in civilian practice. The etiologic factor of emotional strain is the most difficult to control, in view of the fact that a large percentage with cardiospasm, diarrhea of undetermined origin, chronic ulcerative colitis and peptic ulcer have the same basic background of emotional instability. Why this group develops these wide divergence of conditions is not clear. In private practice I have been impressed by a careful searching history, confirmed

by x-ray, by the high indications of peptic ulcer in the teen age period. I do not know of any one better equipped than Commanders Twiss and Parsonnet to state that these patients with peptic ulcer should not be admitted to the armed forces and that, if they should enter the service and develop ulcers, early separation from service be recommended. On the other hand, in private practice these patients will have to be adjusted to continue to earn a living and carry on as near as possible the normal functions in the highly competitive field of living. We may be able to suggest change of occupation, but here again one is disturbed by the finding of peptic ulcer in the most diverse types of work, and it narrows it down to the fundamental discussion of the individual with the ulcer and the efforts of the private practitioner who sees these patients early to emphasize the advisibility of early, properly controlled treatment. At the present moment those who are interested in the care of peptic ulcer are advocating a form of therapy that can be carried out by every practitioner utilizing a small group of medicinal agents, a generous simplified diet and definite personal interest in each patient. Under these circumstances it may be possible to avoid the serious complications of the disease.

CAPT. J. R. TWISS (MC), U.S.N.R.: We are entirely in agreement with the conclusion of Dr. Jordan relative to the importance of the recognition of patients having an ulcer diathesis and instituting, as soon as possible, active treatment as well as prophylactic treatment for the purpose of preventing recurrences as far as this is possible. The prompt breakdown of patients who have previously had ulcer, after coming into service, is due in no small measure to the complete inability of the medical officer to control conditions for the purpose of preventing recurrent ulcer symptoms. We are, furthermore, in full agreement with the proposal of Colonel Kantor that all patients giving a history suggestive of ulcer should be fully investigated before being admitted to the armed forces. This procedure would relieve the government of a tremendous and unnecessary financial liability in the treatment of these patients, who are of no actual value to the service after having been accepted. Our objective in regard to the retention of experienced men who desire to remain in the service, as far as circumstances permit, is in accordance with the policy expressed by Colonel Kantor. Captain Snell has shown a very practical grasp of the problems with which we have had to contend, especially in his recognition of the fact that the patient with peptic ulcer tends to utilize his knowledge of having this condition as an admitted means to obtain a discharge from the service. We feel that all patients admitted with suggestive symptoms or with a diagnosis of gastrointestinal disease should have the benefit of a gastrointestinal series. In those having no positive x-ray evidence, the diagnosis of peptic ulcer is never made unless the history and other diagnostic findings provide what we consider incontestable evidence of this condition. Dr. Aaron's contention that in most young people the symptoms of nausea and vomiting are of functional origin is well borne out by our series of patients, in that practically none showed x-ray evidence or operative findings of obstruction sufficient to account for these symptoms.

Poliomyelitis.—Despite thirty years of concentrated clinical, epidemiological and experimental study, poliomyelitis remains as an outstanding challenge to preventive medicine. No means of preventing or curing the acute stage of the disease is yet in sight. There is equally no adequate knowledge on which to base any straightforward account of the natural history of the disease. . . . Poliomyelitis, in its clinically visible form of fever and signs of central nervous system infection followed by more or less extensive paralysis, is known to be due to invasion by one of the smallest viruses. The virus can produce essentially similar infections in several species of monkey but, except for a small proportion of strains, in no lower type of animal. The more important characteristics of the virus and the pathology of experimental infections have been fully elucidated, but a great deal remains to be learnt in regard to the epidemiology of the disease.—Burnet, Frank MacFarlane: Virus as Organism, Cambridge, Mass., Harvard University Press, 1945.

TREATMENT OF DIABETIC ACIDOSIS AND DIABETIC COMA

THOMAS P. ALMY, M.D.
KATHARINE SWIFT, M.D.
AND
EDWARD TOLSTOI, M.D.
NEW YORK

Although the agents available for the treatment of diabetic acidosis have remained the same for many years, there is today no universally accepted procedure in the management of this common medical emergency. No one will deny that the two most urgently needed substances are insulin and isotonic solution of sodium chloride, but worthy of critical review are (*a*) the desirability of administering glucose in the early phases of therapy, (*b*) the need for frequent determinations of blood sugar and carbon dioxide combining power in regulating the treatment, (*c*) the optimum size of the doses of insulin, and their frequency of administration, and (*d*) the need for the constant attendance of a team of specialists in the field of diabetes mellitus to guide the therapy.

At the New York Hospital, during the past five years, nearly all patients with diabetic acidosis and diabetic coma have received liberal amounts of glucose by mouth, by vein or by both routes from the time of admission. The course of therapy has been followed almost entirely by clinical signs and by qualitative tests for sugar, acetone and diacetic acid in the urine. The blood sugar and carbon dioxide combining power have rarely been used as indexes of the severity of the disorder or of the progress of treatment, and indeed these estimations have been made, in most instances, only after recovery from ketosis. The individual insulin doses have been small and repeated frequently. All but a few of our cases have been treated by the intern, with advice from the medical resident physician. As these practices differ considerably from those advocated by such authorities as Joslin [1] and Root,[2] we have been led to review the results of our therapy. The facts obtained are the basis of this report.

METHOD OF TREATMENT

Interns are instructed to treat diabetic acidosis and diabetic coma as follows:

1. As soon as the diagnosis is made, give 25 units of regular insulin subcutaneously and repeat this every half hour until the patient is free from all signs of ketosis, as judged by clinical and laboratory evidence.

2. When possible, replacements of fluid, salt and carbohydrate should be given by mouth. Give in each hour (*a*) 50 to 75 Gm. of glucose as orange juice (500-750 cc.), grape juice (300-450 cc.) or fortified fruit juices; (*b*) salty broth, 200 cc.; (*c*) enough water to make the total fluid intake 1,000-1,500 cc.

3. If the patient is drowsy or comatose, or if the patient has vomited or has been nauseated during the preceding twenty-four hours, therapy must be begun by the intravenous route: (*a*) Infusion of 5 per cent glucose in isotonic solution of sodium chloride, which should run in at first at the rate of 750-1,000 cc. per hour. (*b*) The rate of this infusion should be reduced

From the Department of Medicine, New York Hospital, and Cornell University Medical College.

1. Joslin, E. P.: Medical Progress, New England J. Med. **232**: 219 (Feb. 22) 1945.

2. Root, H. F.: The Use of Insulin and the Abuse of Glucose in the Treatment of Diabetic Coma, J. A. M. A. **127**: 557 (March 10) 1945.

as soon as dehydration is relieved. Patients over 40 years of age, any with notable arteriosclerosis and any with cardiovascular disease may have to be infused at slower rates and should be observed frequently and with extreme care for signs of increased venous pressure and passive congestion of the lungs.

TABLE 1.—*Age Distribution*

Age	Number of Patients
0 - 10	3
11 - 20	16
21 - 30	18
31 - 40	12
41 - 50	7
51 - 60	10
61 - 70	3
71 and over	2

TABLE 2.—*Duration of Diabetes*

Not previously diagnosed	13
Under 1 year	9
1 to 5 years	44
Over 5 years	27

Return to the oral route as soon as the patient is fully conscious and free from nausea.

4. Test a urine specimen every half hour or every hour for sugar, acetone and diacetic acid. (Catheterize if necessary.) Continue the foregoing therapy until the urine is free from acetone and diacetic acid. Do not leave the ward until this is accomplished.

5. When ketonuria and all clinical signs of acidosis have disappeared, give fruit juice and 25 units of insulin at two hour intervals for at least eight more hours.

6. If the patient is anuric, guide the therapy by frequent estimations of the carbon dioxide combining power of the blood. This rule will also apply if acidosis should occur without the presence of acetone bodies in the urine. (Such cases are exceedingly rare and have not yet occurred in our experience.)

Every patient whose urine contains sugar and acetone bodies is treated thus, regardless of the severity of the diabetic acidosis. Many of the milder cases so treated have been excluded from the series reported here, but no fatal case has been excluded, nor any in which the acidosis did not promptly respond to treatment.

TABLE 3.—*Daily Insulin Requirements*

Units of Insulin	Number of Cases
None	5
Less than 25	13
25 to 50	32
More than 50	26

TABLE 4.—*Precipitating Causes of Diabetic Acidosis*

Breaking diet		27
Eating more	2	
Eating less	25	
Failure to take insulin		44*
Infection		46
Severe	22	
Mild	24	
Coronary occlusion		1
Fracture of femur, toxic nodular goiter		1
Insulin resistance		1
None observed		15

* This figure includes (*a*) neglect of insulin administration, sometimes due to misunderstanding, (*b*) cases in which diabetes had not been previously diagnosed and (*c*) those who had not previously been thought to require insulin.

This report concerns all cases of diabetic acidosis and diabetic coma admitted to the New York Hospital from Jan. 1, 1940 to Dec. 31, 1944 which satisfied the following criteria: 1. Ketonuria, determined by the ferric chloride reaction for diacetic acid and/or the nitroprusside test for acetone. The test for acetone was read as 3 plus or 4 plus in all but 5 cases, in which a 2 plus reaction was associated with severe clinical signs of acidosis. 2. One or more clinical signs of acidosis, as listed in table 5. 3. The requirement of at least 75 units of insulin over a period of three hours to eliminate ketonuria.

There were 99 such occurrences in 71 patients: 28 of the patients were men and 43 were women. The age distribution is shown in table 1. Twenty-two, or 31 per cent of the patients, were over 40 years of age.

The duration of diabetes prior to admission for diabetic acidosis is shown in table 2. In 13 instances the existence of diabetes was not previously suspected, and in 27 instances the patient had been treated for diabetes for five years or more. This distribution is similar to that reported by Joslin.[3]

In table 3 are summarized the daily insulin requirements of these patients before or after hospitalization. These amounts of insulin were sufficient to produce good clinical control by our criteria, namely maintenance of weight, freedom from symptoms of diabetes, freedom from ketosis and absence of insulin reactions. We are impressed that the "mildness" of diabetes, as indicated by the need for little or no insulin, is not a guaranty against acidosis.

TABLE 5.—*Clinical Symptoms and Signs*

		Not Noted
Mental status		5
Conscious	42	
Semiconscious	49	
Comatose	3*	
Nausea	38	4
Nausea and vomiting	31	4
Kussmaul breathing	19	18
Acetone breath	63	26
Soft eyeballs	44	35
Anuria and circulatory shock	1	

* One additional patient was not in coma on admission but died in coma. See fatal case 7.

In table 4 are listed the precipitating causes of diabetic acidosis in these patients. It is confirmed here that infection and omission of insulin are the most important causes of acidosis. On evaluating the dietary intake over the last few days before admission, it was found that 25 patients had eaten less than their usual diet and that only 2 had eaten more. One of these 2 patients had run out of insulin the day before admission. The other patient had stopped taking insulin two days before admission and during this period had eaten her usual diet plus a large quantity of peanuts. In most publications, one of the important causes of ketosis in a diabetic patient is given as "dietary indiscretion," and this is often interpreted as eating concentrated sweets or other foods in addition to the permitted diet. After our experience we find it difficult to accept the dictum that overeating per se is a cause of diabetic acidosis.

The clinical signs and symptoms exhibited by these patients are presented in table 5. Coma is defined by us as a state of unconsciousness in which the patient fails to respond to any stimulus, and thus the 3 patients

3. Joslin, E. P.; Root, H. F.; White, Priscilla; Marble, Alexander, and Hunt, H. M.: Diabetic Coma, M. Clin. North America **16**: 791 (Jan.) 1933.

in that category belong in "stage 4" of coma as defined by Rabinowitch.[4] All cases in which the condition was described as drowsy, semicomatose or in other ways indicating impairment of the sensorium with some responsiveness are here listed as semiconscious. Thus slightly over half of the patients had disturbances of the sensorium and fall into the various categories of coma outlined by Rabinowitch.[4] Only 1 patient was in circulatory shock, and this patient only developed anuria. In 46 of the cases there was an infection in association with diabetic acidosis, and in 22 it was so severe as to constitute by itself an indication for hospitalization.

The total amounts of insulin, glucose, sodium chloride and fluid administered in the first twenty-four hours are given in table 6. Only 2 patients received single insulin doses greater than 50 units, and 1 of these was a case of proved insulin resistance (fatal case 7). A few of the patients received protamine zinc insulin during the first day in the hospital, but in general it has been our custom to postpone the use of slower acting insulin until at least two days after entry. In 27 of the cases, sixth molar sodium lactate was given intravenously.

The interval required to relieve ketosis was estimated from the time of disappearance of ketonuria. This

TABLE 6.—*Quantities of Therapeutic Agents Given in First Twenty-Four Hours*

	Minimum	Maximum	Median
Insulin units	75	885	187
Glucose, Gm., intravenous	10	750	112
Glucose, Gm., total, all routes	50	800	312
Sodium chloride, Gm., intravenous	4.5	55	15.5
Fluid, cc., all routes	1,500	17,700	5,250

could be determined accurately in 95 cases. From table 7 it can be seen that 20 of these (21 per cent) remained in ketosis longer than twelve hours.

Table 7 reveals that the time of recovery was about the same irrespective of the route of administration of glucose. The patients in whom recovery was delayed did not receive more glucose than those who recovered more rapidly: The median intake of glucose in the first twenty-four hours by these patients is exactly the same (312 Gm.) as that reported for the entire series in table 6. The patients in whom recovery was delayed beyond twelve hours did, however, receive less insulin in each hour than is desirable. Although we recommend a constant dosage of 50 units of insulin per hour until recovery, only 1 of these patients received that amount. The rest received from 4 to 27 units per hour, with a mean dosage of 12 units per hour. We are led to conclude that delay in recovery was associated not with the excessive intake of glucose but with inadequate dosage of insulin.

There were 7 deaths in this series. The salient features are presented in table 8, and the protocols are given herewith:

CASE 1.—A man aged 55 was admitted with coronary occlusion and mild diabetic ketosis. He was completely conscious throughout. The ketosis was cleared in eight hours with 130 units of insulin, oral glucose, oral and parenteral fluids and salt in limited quantities. There was no ketosis in the ensuing twenty-eight hours, until death. Autopsy showed two fresh myocardial infarcts, with aneurysm formation and beginning rupture of the ventricle.

4. Rabinowitch, I. M.; Fowler, A. F., and Bensley, E. H.: Diabetic Coma, Ann. Int. Med. **12**: 1403 (March) 1939.

CASE 2.—A woman aged 49 had acute purulent frontal sinusitis due to hemolytic Staphylococcus aureus, with cellulitis of the face, left exophthalmos and ophthalmoplegia. Her urine on admission contained 2 plus acetone and 2 plus diacetic acid, and the patient was drowsy. She was cleared of ketosis in eight hours, using 125 units of insulin plus intravenous sodium lactate and oral and intravenous glucose. Despite this therapy

TABLE 7.—*Duration of Ketonuria*

	Oral Glucose Only		Oral and Intravenous Glucose		All Cases	
Time	Number	Per Cent	Number	Per Cent	Number	Per Cent
Under 4 hrs.	4	14	4	6	8	8
4 to 12 hrs.	17	61	50	75	67	71
Over 12 hrs.	7	25	13	19	20	21
Total	28	100	67	100	95	100

her mental state did not improve, and she later became completely comatose. Lumbar puncture showed turbid spinal fluid with 150 white blood cells per cubic millimeter. Ketosis did not reappear in the remaining forty-eight hours of her life, although the temperature remained between 39 and 40 C. (102.2-104.F.). She had massive edema of the orbits and face, unrelieved by surgical drainage of the frontal and ethmoid sinuses, together with the removal of necrotic frontal bone. The patient was further explored through burrholes by the neurosurgeon, who found no localized intracranial abscess and felt that she had a cavernous sinus thrombosis. Despite adequate penicillin therapy she died on the third day in the hospital.

CASE 3.—A woman aged 40 was admitted with cellulitis of the neck and severe diabetic acidosis. She was semicomatose. In another service of our hospital, to which she was admitted, ketosis was cleared only after twenty to twenty-four hours, intravenous glucose and sodium lactate being used and only 160 units of insulin. She then relapsed into acidosis, which was finally cleared forty-five hours after admission and twenty-

TABLE 8.—*Fatal Cases*

Case, Sex, Age	Mental Status	Duration of Ketosis	Interval Free of Ketosis Before Death	Associated Conditions	Autopsy
1 ♂ 55	Conscious	8 hrs.	28 hrs.	Myocardial infarcts	Yes
2 ♀ 49	Semiconscious	8 hrs.	48 hrs.	Sinusitis, cellulitis, probable cavernous sinus thrombosis	No
3 ♀ 40	Semiconscious	45 hrs.	22 hrs.	Staphylococcic septicemia and pneumonia	Yes
4 ♂ 43	Conscious	6 hrs.	4 days	Cirrhosis; type 1 pneumococcic pneumonia with bacteremia	Yes
5 ♀ 77	Semiconscious	14 hrs.	8 days	Fractured neck of femur, generalized atheromatosis; ? thyrotoxicosis	Yes
6 ♀ 53	Conscious	2 hrs.	0	Lobar pneumonia	No
7 ♀ 50	Comatose	48 hrs.	0	Insulin resistance	Yes

two hours prior to death. During this time the temperature fluctuated from 39 to 41 C. (102.2-105.8 F.). The patient developed pneumonia and septicemia; both blood and sputum cultures were positive for hemolytic Staphylococcus aureus, the blood culture showing 12 colonies per cubic centimeter. At this time she was transferred to our service, but she died seven hours later in pulmonary edema. Autopsy showed carbuncle of the neck, septic infarcts of the lungs, bronchopneumonia of all lobes and bilateral empyema, all due to the hemolytic Staphylococcus aureus.

Case 4.—A man aged 43 was admitted in diabetic ketosis, lobar pneumonia and bacteremia due to Pneumococcus type 1. The blood culture showed 260 colonies per cubic centimeter. Ketosis was mild and was cleared in six hours, only 75 units of insulin being used with oral and intravenous glucose. However, occasional traces of acetone reappeared until death. Despite the use of sulfadiazine and 200,000 units of type 1 antipneumococcus serum, the blood culture on the third hospital day showed 250 colonies per cubic centimeter and there was clinical evidence of spread of the pneumonic process. The patient died on the fourth day in the hospital, and autopsy showed complete consolidation of the right lower and right middle lobes, nearly complete consolidation of the right upper lobe, and patchy involvement of the left lung. In addition there were Laënnec's cirrhosis of the liver, with splenomegaly and subarachnoid hemorrhage.

Case 5.—A woman aged 77 was admitted with fracture of the neck of the femur and diabetic acidosis. She was drowsy on admission, and ketosis was cleared in fourteen hours by means of 175 units of insulin and oral and intravenous glucose. There was no ketosis in the eight days prior to death, which was unexplained even after autopsy. The findings at autopsy were fracture of the neck of the femur, generalized atheromatosis, nephrosclerosis, slight hyperplasia of the thyroid gland and slight bronchopneumonia.

Case 6.—A woman aged 53 was admitted with lobar pneumonia of about seven days' duration, then involving almost all of the right lung. She was alert on admission but showed a heavy ketonuria. She received 90 units of insulin, 1,000 cc. of 10 per cent glucose in saline solution intravenously, plus oral glucose, but remained in ketosis until death two hours later in pulmonary edema.

Case 7.—A diabetic woman aged 50 was admitted for study of insulin resistance. While in another hospital she had continued to show acetone and glucose in the urine even while receiving 400 units of insulin per day. An insulin tolerance test at that time had shown no response of the blood sugar to 50 units of regular insulin. On admission to our hospital she was alert and in mild ketosis, which cleared in fifteen hours on oral feeding and 625 units of insulin. On her fifth day in the hospital she again had asymptomatic acidosis, which cleared in twelve hours with 430 units of insulin, oral glucose and intravenous glucose. She was then free of ketosis until the tenth day in the hospital, after which progressively severe acidosis developed and she died in coma three days later. The administration of insulin was complicated by the development of local inflammation at the sites of injection; much of her insulin was given intravenously, but it was not more effective by this route. In the last twenty-four hours she received 1,310 units of insulin and 400 Gm. of glucose intravenously. Autopsy revealed no other cause of death. The patient's serum was shown to protect mice against convulsive and fatal doses of insulin.

Of these 7 fatal cases, only 2 remained in ketosis until the time of death. The others were freed from ketosis from one to eight days before death. Three of the patients were never drowsy or comatose. In 6 cases death was directly due to a highly fatal associated disease, not to diabetic acidosis, and in 4 of these our conclusions were substantiated by postmortem examination. In 1 of these cases (case 3) the fatal dissemination of a staphylococcic infection occurred after delay in the vigorous treatment of acidosis. This patient was transferred to our service in a moribund condition seven hours before death. In the seventh case the cause of death was diabetic coma, and it was established that this patient had specific neutralizing antibodies for insulin. We conclude that the crude mortality rate in this series is 7 per cent and that the mortality rate from diabetic acidosis is 1 per cent.

COMMENT

These results of therapy are not presented for purposes of comparison with those obtained at other hospitals where different methods of treatment are used. Such comparisons are not valid because of the many factors which determine the prognosis in the individual case. These factors have been exhaustively studied and discussed by Rabinowitch,[4] and the more important ones from our experience are (*a*) the chronological and physiologic age of the patient, (*b*) the presence of associated diseases of serious import and (*c*) the severity of diabetic acidosis.

In 819 cases from four separate clinics, Rabinowitch[4] found a regular increase in mortality rate with advancing age. In patients under 20 years of age the mortality rate was 5.1 per cent. About one third of the patients were over 40 years of age, and in these the mortality rate was 48.5 per cent. One third of our patients were 40 years of age or over, and all of the fatal cases were in this age group.

The mortality rate may be expected to be higher when to the risk of death from diabetic acidosis is added the risk of death from some other dangerous disease. In almost all reported series of cases of diabetic acidosis the mortality in complicated cases far exceeds that in uncomplicated ones. Indeed, there should be almost no mortality from uncomplicated diabetic acidosis under present day management. All but 1 of our fatal cases were complicated by another highly fatal disease which appeared on good clinical or pathologic grounds to be the major cause of death.

The severity of diabetic acidosis is estimated in some clinics largely by the state of consciousness of the patient, in others largely by the carbon dioxide combining power of the plasma. Rabinowitch[4] has clearcut evidence on the relative prognostic value of these indexes. There was a direct relationship in his 125 patients between the degree of unconsciousness and the mortality rate. But in 713 collected cases there was no relationship between the level of the carbon dioxide combining power and the mortality rate. The highest mortality rate, in fact, occurred in patients with a carbon dioxide combining power above 25 volumes per cent! It has therefore seemed better to rely on the state of consciousness of the patient as the more correct and convenient index of the degree of ketoacidosis.

Although this series contains an average proportion of elderly patients and numerous instances of serious associated diseases, only a minority of our patients had serious disturbances of the sensorium.[5] It may be argued that our method would appear less successful if we had had more comatose patients. Yet the conscious and the unconscious patients in this series were handled with equal ease, and the difficulties encountered were those of complicating conditions rather than those of intractable ketosis. In our hands this method of therapy has produced results as good as those achieved by other methods in current use. Before recommending it for general use, we would ask (*a*) Is it rational? (*b*) Is it practical, convenient and safe?

A patient in diabetic acidosis has invariably lost in his urine during the preceding days large amounts of water, sodium and glucose. This has been associated with a relative insufficiency of insulin. Particularly when nausea and vomiting are present, these losses

5. This is probably due in part to the fact that our hospital does not maintain an ambulance service. Hence most patients who are found in an unconscious state in our neighborhood are taken to city hospitals.

are not compensated by increased intake. The loss of sodium and water from the extracellular fluid is adjusted by transfer of water and potassium ion from the intracellular fluid. The loss of glucose is compensated by breakdown of liver glycogen, by gluconeogenesis from protein and by substituting fat for carbohydrate as a primary source of energy. Fat is burned for energy after being first broken down by the liver into ketone bodies. All of these processes are efficient only within limits, and, as Stadie [6] and Mirsky [7] have shown, when the rate of production of ketones by the liver exceeds the maximal rate of utilization of ketones by the tissues, ketones accumulate in the blood, combine with fixed base, and produce acidosis and ketonuria. The fundamental disturbance in diabetic acidosis is a depletion of water, sodium and glucose in association with a relative insufficiency of insulin.

There is general agreement that the most important step in the therapy of acidosis is the use of large quantities of insulin, and to this we subscribe. There is likewise general recognition of the need for restoring water and sodium ion, and in all clinics the patients receive water and sodium chloride, by vein if necessary. Sodium chloride is useful for relieving acidosis only if the kidney is capable of excreting the chloride in combination with ammonium ion, the sodium being retained as base. If the kidney is insufficient, most will agree that it is better to supply sodium as sodium lactate or sodium bicarbonate.

There has been disagreement concerning the use of glucose in the treatment of diabetic acidosis and diabetic coma. The state of depletion of carbohyrate in the body in diabetic coma has not generally been recognized because the blood, the only tissue tested under such circumstances, always shows an excessive concentration of sugar. That there is truly a state of depletion of carbohydrate in the body in coma is attested by the figures of Soskin,[8] although these figures are contested by Root.[2] The technical difficulties of obtaining accurate analyses for glycogen on human patients are almost insuperable, and, rather than rely on data of questionable accuracy to determine whether the body is depleted of sugar, we have chosen to recognize the simple fact that there is an extra loss of carbohydrate from the body with unchanged or with diminished intake. In other words, the body is in negative carbohydrate balance.

It has been alleged by some writers that glucose "neutralizes" the action of insulin in the treatment of diabetic acidosis. This thesis has been supported almost entirely by reports of cases in which death has followed the use of glucose in the treatment of ketosis. In these cases it has been impossible to establish a clear relation of cause and effect. Let us inquire how this thesis fits in with what is known of the physiology of diabetic acidosis and of the actions of insulin:

The central problem in the relief of diabetic acidosis is to suppress the formation of ketone bodies in the liver. This is probably accomplished both by stimulating carbohydrate oxidation and by inducing the accumulation of glycogen in the liver. It is well known that insulin favors the accumulation of liver glycogen, and this is thought to be due to an inhibition of glycogenolysis. Banting and his associates [9] demonstrated that in fasting animals the hyperglycemia which follows the administration of epinephrine is prevented by insulin. Does glucose oppose the accumulation of liver glycogen in the diabetic organism? It does not. Increases of liver glycogen in diabetic animals have been obtained by Major and Mann [10] and by Bodo and his associates [11] by infusions of glucose without the use of insulin. Mirsky [7] has shown that diabetic patients in ketosis, either deprived of insulin or receiving suboptimal doses of insulin, exhibit diminished ketonuria and greater retention of carbohydrate when the intake of carbohydrate is greatly increased. In their effects on the level of liver glycogen, glucose and insulin are not antagonists. It seems more logical to regard them as substrate and enzyme in the same chemical reaction. This is in line with the findings of Macleod,[12] who studied the effects of insulin on diabetic animals with various levels of glucose intake, and concluded that:

> The action of insulin on glucose metabolism no doubt proceeds according to the same general laws as govern enzyme action, namely that the amount of substrate which is acted on is not a linear function of the amount of enzyme but is relatively much greater when there is a large excess of substrate compared with the amount of enzyme. This latter condition is fulfilled when small amounts of insulin are given, or when, with larger amounts, the intake of sugar is increased.

Campbell, in the clinical section of the same paper, put this into practical terms. Speaking of the treatment of diabetic acidosis,

> It has been the custom in Toronto [he said] to use 1 gram of glucose or other carbohydrate for each unit of insulin.

Indeed, the only known antagonism of glucose and insulin in diabetic individuals is in relation to the level of blood sugar. How unimportant a normal level of blood sugar is in recovery from diabetic acidosis is indicated by the fact that almost every one of our cases was cleared of acidosis while having a 4 plus glycosuria.

We differ from some other students of this problem in the degree of emphasis we place on the determination of the blood sugar and the carbon dioxide combining power in gaging the severity of acidosis and the progress of therapy. We have already indicated of how little value is the carbon dioxide combining power in determining the prognosis. We do not deny that a well run determination by an experienced technician is an accurate index of the severity of acidosis at the time it is taken, but though it may outweigh in accuracy the impression one gains from observing the respirations and the state of consciousness of the patient, this advantage is overcome by the delay in receiving the report. We have found that the result of such a determination is not available until from one to three hours later, and in many instances it is then only of academic interest. The determination of blood sugar is of no value for guiding therapy when glucose is administered as a part of the treatment. Our policy with respect to the use of blood chemical determinations is to use them when it is convenient to do so and to regard them as supplementary aids but almost never to perform them as emergency procedures.

6. Stadie, W. C.: Fat Metabolism in Diabetes Mellitus, Ann. Int. Med. **15**: 783 (Nov.) 1941.
7. Mirsky, I. A.: The Etiology of Diabetic Acidosis, Proc. Am. Diabetes A. **1**: 51, 1941; J. A. M. A. **118**: 690 (Feb. 28) 1942.
8. Soskin, Samuel: The Storage and Significance of Tissue Glycogen in Health and Disease, Proc. Am. Diabetes A. **2**: 119, 1942.
9. Banting, F. G.; Best, C. H.; Collip, J. B.; Macleod, J. J. R., and Noble, E. C.: The Effects of Insulin on Experimental Hyperglycemia in Rabbits, Am. J. Physiol. **62**: 559 (Nov.) 1922.
10. Major, S. G., and Mann, F. C.: The Formation of Glycogen Following Pancreatectomy, Am. J. Physiol. **102**: 409 (Nov.) 1932.
11. Bodo, R. C.; Co Tui, F., and Farber, L. A.: The Relation of Insulin to Liver Glycogen, Am. J. Physiol. **101**: 10, 1932.
12. Campbell, W. R., and Macleod, J. J. R.: Insulin, Medicine **3**: 195 (Aug.) 1924.

The need for laboratory aids in estimating the severity of acidosis is further minimized in our method by the fact that we do not individualize the dosage of insulin but instead treat each patient with large amounts of insulin until ketosis is cleared. The individual dose of insulin given is small (25 units) but it is repeated every half hour. Thus it is intended that the patient receive in a period of six hours 300 units of insulin, a total dosage which is similar to that given in most clinics over an equal period of time. We believe this practice is effective because patients with all degrees of acidosis have responded equally well, and the only patient who failed to respond was one who was shown to be insulin resistant. We believe it is safe because we simultaneously administer glucose, and this has prevented the occurrence of hypoglycemia. A further danger of too vigorous treatment of acidosis has recently been suggested by Root, who points to the possibility of overloading the liver cells with glycogen. This hypothesis has been based, as far as we know, entirely on histologic studies of the liver. Such studies are not noted for their accuracy. Ketone production in the liver is probably suppressed at a time when the concentration of glycogen in the liver is still far below normal. Then when the ketone bodies disappear from the urine the dosages of insulin and glucose are reduced. In these circumstances it seems unlikely that there is danger of damaging the liver by excessive deposition of glycogen.

In most clinics it is the rule to administer large doses of insulin at a time, yet there are hardly two clinics that have the same concept of optimal dosage. The maximal rate of utilization of insulin is not known, and all routines for its use have been empirically constructed. In our plan of therapy, small doses of insulin are given and repeated frequently. We think that such a course is more efficient, because it has been known since the original work of Banting and his associates that the law of diminishing returns applies to insulin: Small doses metabolize more carbohydrate per unit than do large doses. We think that this procedure is safer, because there is less likelihood of severe hypoglycemia from the delayed effect of large doses of insulin.

We are aware that as good results as ours in the treatment of diabetic acidosis have been achieved by workers with long experience in this field, using a method which forbids the use of exogenous glucose, which demands control of the progress of the patient by frequent chemical testing of the blood and which relies on the empirical selection of proper doses of insulin. We are inclined to regard these good results as due in large part to the great experience of these physicians, for we know of other groups with equal medical wisdom and conscientiousness who have failed to achieve equal results by the same method. Our method has not been used extensively by specialists in diabetes mellitus; the results reported here were achieved by interns. We are convinced that this method is simpler. It allows the treatment of all cases of diabetic acidosis with equally large individual doses of insulin, without fear of insulin reactions. It does not require the use of time consuming laboratory tests. It does not require the constant attendance of a diabetes specialist to select the appropriate dose of insulin. In other words, this method can be applied by any physician who has facilities for testing the urine and who understands the basic functional pathology of diabetic acidosis.

SUMMARY

1. A method of treatment of diabetic acidosis has been adopted in which are used small and frequently repeated doses of insulin and replacement of water, sodium chloride and glucose by mouth or by vein.

2. The course of therapy is guided by clinical signs and qualitative analysis of the urine for sugar and acetone bodies.

3. In the great majority of cases at our institution, treatment is given by the house staff, guided only by the resident physician.

4. Among 99 cases of diabetic acidosis and diabetic coma so treated during the past five years there were 7 deaths, only 1 of which was due to diabetic acidosis per se. In that patient there was proved insulin resistance of a high order.

CONCLUSION

Our experience indicates that patients with diabetic acidosis or diabetic coma can be successfully treated with small, standard and frequently repeated doses of insulin, that the administration of glucose in this disorder is beneficial and not harmful, that it is not necessary to follow the blood sugar and carbon dioxide combining power as guides to therapy, and that with such a simple method the constant attendance of specialists in diabetes mellitus is, in all but rare instances, unnecessary.

Clinical Notes, Suggestions and New Instruments

BUERGER'S DISEASE IN A BOY OF SIXTEEN YEARS

CAPTAIN EDISON D. FISHER
MEDICAL CORPS, ARMY OF THE UNITED STATES

Thromboangiitis obliterans usually affects males between the ages of 20 and 50 years. Horton [1] reported a series of 984 cases seen over a period of thirty years at the Mayo Clinic in which their youngest patient was 17 years of age. A search indicates that the subject of the present report is the youngest in the English literature. The disease developed while he was 15 and was well advanced when we saw him shortly after his sixteenth birthday.

REPORT OF CASE

J. D. H., a high school student aged 16, presented himself to the outpatient department of the Mason Memorial Hospital at Murray, Ky., on Dec. 13, 1938 because of pain and tenderness in his right foot. Four months previously he had observed that the ball of this foot was tender. In the course of time this tenderness was replaced by numbness, then a burning sensation and finally severe pain, especially in the fourth and fifth toes. The right calf was sore and cramped when he walked. The situation became progressively worse, so that on December 26 he was admitted to the hospital for treatment. He arrived about midnight complaining of excruciating pain in his right foot, especially the fifth toe.

His ancestors, who had been Americans for several generations, were of Scotch-Irish extraction with nothing of special significance in the family history. He was a normal breast fed infant who had passed through a normal childhood with no incidents of clinical interest other than uncomplicated measles, whooping cough and scarlet fever. His adolescence had been uneventful until the onset of the present disability. He had smoked cigarets for about two years and at the time of admission was using about twenty cigarets a day.

Physically he was rather slender and evidently rapidly growing, and his general development was about normal. He did not appear acutely ill but evidently was suffering much pain.

1. Horton, B. T.: The Outlook in Thromboangiitis Obliterans, J. A. M. A. **111**: 2184-2189 (Dec. 10) 1938.

His temperature was 98 F., pulse rate 84, respiratory rate 16 and blood pressure 126/74. His height was 68 inches (173 cm.) and weight 122 pounds (55 Kg.). A rather extensive chronic acne was present on the face and upper part of the chest. No disease or abnormality of the heart or lungs was found. The upper extremities were normal with no disturbances of blood supply, trophic changes or signs of autonomic imbalance.

No other significant departures from normal were present aside from the findings in the right lower extremity. The right foot was cold, mottled and cyanotic. A gangrenous appearing ulcer on the fifth toe extended from the base of the nail to the medial aspect of the toe. The ball of the foot and the fourth and fifth toes were very tender and painful. Pulsations were absent in the dorsalis pedis and posterior tibial arteries and reduced in volume in the popliteal artery. When the foot was elevated it became blanched, when dependent it was very cyanotic and when on the horizontal plane its color was a mottled cyanosis. The right leg below the midcalf was constantly colder than the left. None of the abnormal findings of the right lower extremity were present on the left.

Mentally he was of average intelligence, well adjusted and emotionally stable. Neurologic findings were within normal limits.

The laboratory findings were not striking. Blood examination revealed a normal red cell count, hemoglobin, leukocyte and differential count, blood sugar and nonprotein nitrogen. A mild leukocytosis appeared for a few days when the toe ulcers were the most extensive. Blood sodium chloride was 454 mg. and the Wassermann reaction was negative. The urine was essentially normal.

On the second hospital day he was given a procaine spinal anesthetic as a diagnostic measure. Anesthesia was produced in both lower extremities. The left leg and foot became pink and warm and the dorsalis pedis and posterior tibial pulsations were voluminous, but very little change, if any, was observed in the right foot. It remained cold and cyanotic and its arteries pulseless. These findings, considered in the light of the history and physical findings, substantiated a diagnosis of thromboangiitis obliterans.

Treatment consisted of Buerger's exercises twice a day, contrast leg baths three times a day, local dressings of butesin picrate to the ulcerated toes and analgesics as needed. A heating cradle at about 95 F. was tried for a few hours, but it so increased the pain that it was removed. All tobacco was discontinued.

During the first ten days of hospitalization all of his symptoms became progressively worse. The ulcer on the fifth toe became larger, and a smaller one appeared on the adjacent fourth toe. The pain became so severe that opiates were required and the patient requested that his toes be amputated. On the eleventh day, apparently without reason, the foot became quite comfortable and the ulcers began to heal. Evidently sufficient collateral circulation had developed to permit rapid and uninterrupted healing. Eighteen days later, when the patient was discharged from the hospital, the ulcers were healed and the foot was comfortable.

On returning home the patient was instructed to take extra good care of his feet, to continue with Buerger's exercises and to return periodically for a checkup. Later he returned to high school and drove a truck during vacations. Sustained brisk walking caused mild intermittent claudication, but he had no more serious difficulty. The arterial pulsations in the right foot remained absent to palpation. Early in 1941 he was accepted by the Navy and according to reports late in 1943 he was still carrying on successfully.

SUMMARY AND COMMENT

1. A case of Buerger's disease had its onset at the age of 15 years.

2. Severe disease of the right foot improved rapidly with Buerger's exercises, contrast leg baths and discontinuance of all tobacco. Nearly four years later the patient had no relapses. It is difficult to evaluate the part treatment played in the unexpected rapid improvement in this patient.

3. The interest and significance of this case report is that Buerger's disease may develop so early in life.

TREATMENT OF SEVERE NECROTIC LESIONS

HIGHER PENICILLIN DOSAGE WITH SIMULTANEOUS REGIONAL SYMPATHETIC BLOCK

MEYER NAIDE, M.D., AND ANN SAŸEN, PHILADELPHIA

Gas gangrene has been reported in fairly large numbers in this war. Forty-four cases with 13 deaths and 8 additional amputations in recovered patients were reported by MacLennon [1] in British army casualties in the North African campaign, an incidence of between 6 and 7 per thousand wounded. Twenty cases were seen by Power [2] in 6,000 casualties in the fighting in Normandy. In 16 of these patients there were 10 amputations and 2 deaths.

Despite increase in the potency of antiserums and chemotherapy, local and systemic, results of treatment of gas gangrene are not much better than they were twenty-five years ago.[1] A consideration of the problem makes it obvious that the reason for failure is twofold: There is an inadequacy in blood supply to necrotic tissue and therefore the amount of penicillin reaching this tissue at the usual blood level is completely inadequate. This may explain the failure of chemotherapy in some patients and the present high mortality and amputation rate. Nowhere in the literature have we been able to find a suggestion that much larger doses of penicillin are required when there is reduced blood flow. Power [2] as recently as May 12, 1945 has gone so far as to state that "early reports on penicillin would indicate that it is powerless to stem the progress of gas gangrene."

That gas gangrene occurs in ischemic, necrotic tissue is recognized.[2] That chemotherapy of ischemic tissue is a special problem in itself has not been recognized.

The importance of this problem was brought to our attention by the death of 1 patient with gas gangrene in an ischemic extremity treated with the recommended dose of 100,000 units of penicillin daily, intramuscularly and the recovery without amputation of another patient with a similar infection in an ischemic extremity, but treated with 600,000 units of penicillin daily, intravenously and with two periods of continuous caudal anesthesia for prolonged complete vasodilatation. This led us to the opinion that penicillin is being used in completely inadequate dosage in severe infections in ischemic tissue.

Following is the report of a case of gas gangrene in an ischemic extremity finally treated by us with massive doses of penicillin and caudal anesthesia:

A woman aged 50 was admitted to the University Hospital on April 24, 1945 with a history of diabetes mellitus for seven years. In March 1945, one month before admission to the hospital, she refused to take her daily dose of 30 units of protamine zinc insulin. One week later she noticed that several toes were black on both feet. A week before admission to the hospital she developed fever. The significant physical findings on admission were a temperature of 102 F., pulse rate of 120, a systolic murmur at the apex, foul-smelling gangrene of the second toe on the right foot and the second and third toes on the left foot, with cellulitis of both feet. The dorsalis pedis pulses were present but the posterior tibials were absent. Cataracts had been removed bilaterally. A note was made on the day of admission that bilateral amputation would probably be necessary to control the infection. The white blood cell count was 14,000, the blood sugar 250 mg. per hundred cubic centimeters. The urine was positive for sugar and acetone. Carbon dioxide was 41 volumes per cent.

The patient was started on 100,000 units of penicillin intramuscularly on the day of admission, April 24. On April 27 x-rays showed a large quantity of gas in the soft tissues of the right foot and to a lesser extent in the left, with either osteomyelitis or demineralization of the right second and the left second and third toes. That day the affected toes were amputated and anti-gas serum and tetanus antitoxin were given. The diabetes mellitus was controlled with diet and 80 units of

From the Peripheral Vascular Section of the Edward B. Robinette Foundation, Medical Clinic, Hospital of the University of Pennsylvania.

The cost of the penicillin used in this work was defrayed in part by the Donner Fund for Needy Patients.

1. MacLennon, J. D.: Anaerobic Infections in Tripolitania and Tunisia, Lancet **1**: 656 (Feb. 12) 1944.

2. Power, R. W.: Gas Gangrene with Special Reference to Vascularization of Muscles, Brit. M. J. **1**: 656 (May 12) 1945.

regular insulin. The 100,000 units of penicillin intramuscularly was continued daily. A culture was made on April 27 which disclosed hemolytic streptococci and staphylococci but no clostridia. A smear on April 30 disclosed gram positive rods, and there was gas on palpation in the left foot. An additional incision was made that day for drainage. The penicillin was increased to 200,000 units intramuscularly on April 30. Despite this the temperature remained elevated, the patient was toxic and the lesions were foul. As a patient had been seen in March 1945 with severe ischemia and gas gangrene who died after treatment with 100,000 units of penicillin intramuscularly daily and local surgery, it was suggested that 600,000 units of penicillin be given daily, intravenously. This was done and within twenty-four hours the temperature dropped to a lower level, remaining below 100 F. The patient received anti-gas serum twice more for a total of 60,000 units (40,000 intravenously and 20,000 intramuscularly). On May 11 and 12 continuous caudal anesthesia was instituted for periods of six hours in an effort to increase blood flow through maximal vasodilatation and thus increase the amount of penicillin reaching the infection in the schemic tissue. The presence of pain and anxiety suggested the possibility that peripheral vasoconstriction was reducing the amount of penicillin reaching the infection. The temperature level dropped within twenty-four hours and it remained below 99 F. from May 11 to 20 and was normal thereafter. The right foot healed completely. The left foot is much improved, although additional surgery may still be necessary.

One other case pertinent to the problem is worth mentioning briefly: A man aged 65 came into the hospital with moist necrosis of the tip of the right third toe. An x-ray showed osteomyelitis of the distal phalanx of this toe. Although the patient had occlusion of the right popliteal artery, he did not complain of claudication, the foot was warm, there was no severe pain and it was evident that he had established an adequate collateral circulation. It was decided to use 300,000 units of penicillin daily, intramuscularly. After five days of treatment the necrotic tip became dry, pain subsided completely and the patient was discharged after ten days in the hospital. The feature that permitted successful treatment of this patient with penicillin intramuscularly was evidence of a good collateral circulation and absence of severe pain despite arterial occlusion in the extremity. There is more adequate blood flow to the toes of a foot with arterial occlusion where an adequate collateral circulation has been established and where this is no vasospasm than there is in the toes of some feet with a patent arterial system and reflex vasospasm due to pain, trauma or cold.

COMMENT

Failure to recognize that ordinary doses of penicillin may be inadequate in treating severe infections in ischemic tissue may be a factor in the high mortality and amputation rate in gas gangrene. Kunkel and Stead [3] found that an ischemic foot may have from one third to one tenth of the normal blood supply. It would therefore seem obvious that penicillin dosage must be increased correspondingly, since only a small fraction will reach an ischemic lesion. Fortunately one can do this with penicillin, whereas this is not equally possible with the sulfonamides, which are more toxic in huge doses. Furthermore the intravenous route, using the high doses, is effective in maintaining such a high concentration that even the smaller amount of blood reaching ischemic tissue may have sufficient penicillin to be effective.

The use of massive doses of therapeutic agents in treating symptoms of ischemia is not new. In 1939 one of us [4] pointed out that the problem in arterial occlusive disease is basically that of a block in transportation. It was found that ischemic neuritis could be relieved in some patients by the use of massive doses of thiamine hydrochloride intravenously (100 mg. daily), whereas smaller doses intravenously or subcutaneously were of no value.

Vasodilation methods such as continuous caudal anesthesia for six or eight hours will increase blood flow and thus the amount of effective penicillin. It must be remembered that ischemia of wounds is the result not only of vascular injury but also of reflex spasm of collateral vessels in response to pain and anxiety.

The importance of early surgical removal of necrotic tissue in patients with gas gangrene has been adequately stressed by others. The use of penicillin locally is also indicated, though it is not sufficient in itself. MacLennon [1] and others have pointed out the importance of using large quantities of antiserums intravenously, if possible, his patients averaging 90,000 to 100,000 units.

In 28 of MacLennon's 44 cases there was severe trauma to a major artery. Power [2] has stressed the vascular factor present in all military wounds with gas gangrene seen by him. In 9 of the 16 patients seen by him there was injury to the popliteal artery; in every case amputation had to be performed. He points out that vasodilatation should be encouraged by hot drinks, heat to the uninjured limbs and alcohol. Smoking should be prohibited to avoid reflex vasoconstriction. Heat is contraindicated. Increased penicillin and vasodilator therapy is indicated despite failure to find gas-forming organisms. In only half of MacLennon's cases of gas gangrene were gas-forming organisms found.

CONCLUSIONS

The present recommended dose of penicillin in gas gangrene is inadequate. The reduced blood supply does not permit an effective amount of penicillin to reach ischemic tissue at the usual penicillin blood level.

It is therefore imperative in these lesions to use much larger doses of penicillin, preferably intravenously, to increase greatly the level of penicillin in the blood reaching the necrotic tissue.

It is also important to increase blood flow simultaneously by regional sympathetic ganglion block.

These methods are to be used in conjunction with present adequate local surgical management and with repeated doses of antitoxin intravenously, if possible.

3. Kunkel, P., and Stead, E. A., Jr.: Blood Flow and Vasomotor Reactions in Foot in Health, in Arteriosclerosis and in Thromboangiitis Obliterans, J. Clin. Investigation **17**: 715 (Nov.) 1938.

4. Naide, Meyer: The Use of Vitamin B_1 in Rest Pain of Ischemic Origin, Am. J. M. Sc. **197**: 766 (June) 1939.

Council on Pharmacy and Chemistry

REPORT OF THE COUNCIL

The following statement has been authorized for publication by the Council. AUSTIN SMITH, M.D., *Secretary.*

INFORMATION ON NEW DRUGS

The Council office receives many inquiries concerning the status of drugs newly introduced in commerce and those which are still in the stage of experimental investigation. On the basis of the information which is available in the Council files and that received from consultants to whom the Council may write, these inquiries are answered. However, to give the best information service it is necessary to have the cooperation of those who are responsible for the introduction and distribution of new drugs, and it is important that such information is received as promptly as possible. Manufacturers and others are urged to keep the Council office informed concerning new developments relating to products in which they are interested. At the same time they should of course notify the Council office if there is any restriction on the information, as such restriction will be observed. Ordinarily it is necessary to transmit a digest of the information only when replying to inquiries, but it should be understood that the Council must be free to comment on the supplied data if the occasion arises for such comment; while these comments would aim to be unbiased, they may on occasion be adverse because, for example, of the lack of evidence to support the claims or implied uses. Nevertheless, the end result would be more complete information and a better understanding of the problems involved. Every one participating in the accumulation of this information would benefit. Manufacturers are invited to keep the Council informed; at the same time members of the medical profession and others with similar interests are also invited to make use of the information available in the Council files. Inquiry may be directed simply to the Secretary of the Council on Pharmacy and Chemistry, in care of the American Medical Association, 535 North Dearborn Street, Chicago 10.

Council on Foods and Nutrition

The Council on Foods and Nutrition has authorized publication of the following report.

GEORGE K. ANDERSON, M.D., *Secretary.*

THE EFFECT OF COCOA ON THE UTILIZATION OF DIETARY CALCIUM

H. H. MITCHELL, PH.D.
AND
JANICE M. SMITH, PH.D.
URBANA, ILL.

Cocoa is one of many foods commonly used in American diets that contains moderate amounts of oxalic acid, i. e. 0.5 to 0.6 per cent. On a similar moisture basis, cocoa contains about the same content of oxalic acid [1] as wax beans and gooseberries, slightly more than green beans, carrots and the peelings of citrus fruit, but slightly less than celery stalks. The oxalic acid of foods is capable of forming an extremely insoluble salt with dietary calcium, and for this reason it may impair the utilization of the calcium of foods in the body. Since cocoa and chocolate are used extensively in milk and ice cream, the possibility that the calcium of these dairy products would thus be rendered less available in digestion and metabolism induced Mueller and Cooney of the Massachusetts State College to study the problem with growing rats. The results of this investigation were published about two years ago [2] and have attracted wide interest among nutritionists [3] and particularly within the dairy industry, since chocolate flavors in milk and ice cream have been introduced evidently in response to a consumer demand and may be presumed to increase the consumption of these excellent foods.

The experiments of Mueller and Cooney were designed to compare the retentions of calcium in the bodies of growing rats receiving a diet containing 0.554 per cent of calcium and 0.472 per cent of phosphorus derived from whole milk powder when this diet was fed alone and when it was supplemented with cocoa in the proportion of 0.84 part of the basal diet to 0.16 part of cocoa. Twenty-one pairs of rats were used in addition to 21 litter-mate control rats killed and analyzed at the beginning of the experiment to permit an estimate of the initial stores of calcium. Within each pair of rats the consumption of the basal diet was equalized, while 1 of the pair received the cocoa powder in addition. After five weeks of feeding the rats were killed and analyzed for calcium and phosphorus.

The results of this well conceived and well executed experiment were clearcut in their significance as revealed by statistical analysis. The rats fed the cocoa supplement, although receiving 19 per cent more dry food, 6 per cent more calcium and 30 per cent more phosphorus than the rats on the basal diet only, grew only 89 per cent as fast and deposited in their bodies only 76 per cent as much calcium and 80 per cent as much phosphorus. The effect of the cocoa supplement on growth had been previously shown by Mueller [4] to be traceable probably to its content of tannic substances. The primary effect on mineral utilization was probably on calcium, since (1) calcium was presumably a limiting factor in the diet, (2) calcium limitation will restrict phosphorus utilization because the two minerals are so largely utilized together as a calcium phosphate in bone formation, and (3) the depression of phosphorus retention by cocoa was so nearly the same as that of calcium retention. However, the oxalic acid content of the cocoa could account for only about one third of the observed depression in calcium retention. No clue to the residual causes of this depression was afforded by the data of the experiment or the discussion of them by the authors. It should be noted that the ratio of milk solids to cocoa in the diets of this experiment was about 3.3 to 1, while in chocolate milk it is ordinarily about 13 to 1.

The cocoa that Mueller and Cooney used was a low cost American process cocoa [5] containing 11.23 per cent fat and 5.25 per cent fiber. Cocoas of this description are common at times on the market, being used in the manufacture of bakery goods and for low cost chocolate syrups and confections, though there seems to be a considerable sale of such products for domestic use by certain retail stores throughout the country.

Similar experiments were made on growing rats at the University of Illinois, essentially the same technic being used as that employed by Mueller and Cooney, but three different kinds of cocoa being studied, i. e. the low cost brand used by the Massachusetts investigators (cocoa A), an American process breakfast cocoa (cocoa B) and a Dutch process cocoa (cocoa C). The latter two cocoas were medium cost cocoas containing 22 to 24 per cent of fat and 3.6 to 3.9 per cent of fiber. In the Illinois experiments the cocoas were incorporated into the basal diets at the expense of sugar instead of being added to the diets, and all diets were equalized in fat content. Nine and twelve pairs of rats were used in each of two tests, and pair mates received equal amounts of their respective diets. However, the body weight gains of pair mates were equalized by additions of sugar to the diet of the laggard rat, which was generally the rat receiving cocoa. The experiments were terminated after four to nine weeks of feeding, depending on the rate of growth in each pair, and the carcasses were analyzed for calcium and, in one test, for phosphorus also.

The amount of extra sugar required to maintain equal gains in the cocoa and noncocoa rats, in percentage of the basal food consumed, averaged 18.3 for cocoa A, 6.1 for cocoa B and 4.6 for cocoa C. The difference in this respect between the low cost cocoa A and the higher cost cocoas is highly significant statistically. The calcium retentions of the rats on the three cocoa diets, expressed as percentages of intakes of calcium, showed depressions of 27.7, 14.8 and 18.4 per cent as compared with the control rats receiving no cocoa. Again the detrimental effect of the low cost cocoa A exceeded statistically the effect of the two medium cost cocoas B and C, which were not statistically distinguishable in this respect. The three cocoas contained essentially the same contents of oxalic acid, 0.55, 0.50 and 0.51 per cent respectively. The depressing effect of the B and C (medium cost) cocoas on calcium retention can be entirely accounted for by their contents of oxalic acid, but as in the work of Mueller and Cooney, only about

From the Division of Animal Nutrition, and the Department of Home Economics, University of Illinois.

1. Kohman, E. F.: Oxalic Acid in Foods and Its Behavior and Fate in the Diet, J. Nutrition **18**: 233 (Sept.) 1939.
2. Mueller, W. S., and Cooney, M. R.: The Effect of Cocoa on the Utilization of the Calcium and Phosphorus of Milk, J. Dairy Sc. **26**: 951, 1943.
3. Editorial Review: Availability of Calcium and Phosphorus in Chocolate Milk, Nutrition Rev. **2**: 68 (March) 1944.
4. Mueller, W. S.: The Significance of Tannic Substances and Theobromine in Chocolate Milk, J. Dairy Sc. **25**: 221 (March) 1942.
5. Personal communication to the authors.

one third of the depression in calcium retention induced by the low cost cocoa A can be so explained.

The chemical (and physical) analysis of the three cocoas does not afford any direct explanation for the more harmful effect of cocoa A as compared with cocoas B and C. Besides the chemical data already given, our analysis indicates the percentages given in the accompanying table.

The higher proportion of cacao shell [6] and possibly the lower proportion of fat [7] may be involved in the greater detrimental effect of the low cost cocoa A, with reference both to growth and to calcium utilization.

The experiments on rats at the University of Illinois were carried out in conjunction with a series of experiments [8] on 7 women students (ranging in age from 18 to 22 years), extending over fifty-one four day calcium balance periods. A low calcium basal diet containing an average of 225 mg. of calcium per day and no cocoa was fed alone in thirteen four day periods, supplemented with cocoa in six periods and supplemented with dairy products (milk and ice cream) and cocoa in thirty-two periods. In the latter periods adjustments were made in the basal diet at the expense of low calcium foods to maintain a fairly constant intake of protein and of calories. The cocoa used in these experiments was an American process cocoa of medium cost, manufactured by the same firm and similar in composition to the cocoa B in the rat studies, except for a somewhat lower fat content,

Analysis of Three Cocoas

	Tannic Substances, %	Theobromine, %	Moisture, %	Proportion Not Passing Through a 140 Mesh Sieve, %
Cocoa A.....	11.80	2.10	5.73	11.51
Cocoa B.....	10.57	1.79	4.70	1.12
Cocoa C.....	6.62	1.44	5.20	0.87

20.22 per cent. This cocoa represents approximately the quality normally used in making dairy syrups for the manufacture of chocolate milk and ice cream. In the supplemented diets the calcium intake was raised to about 700 mg. daily, of which dairy products, milk or milk and ice cream, supplied about 71 per cent. The cocoa was added to the diets in amounts varying from 5.5 Gm.[9] to 34.8 Gm. daily and, in the different diet periods, it was incorporated in milk alone, in ice cream, in cookies, in candy or in syrup. For each type of diet with reference to cocoa dosage and manner of incorporation in the foods, each subject during half of the period received cocoa and during the other half of the period received none. Hence, for each of the four diets, comparisons of calcium retention were possible between subjects and between periods on the same subject.

The results of the experiments on human subjects were essentially negative in their significance in the sense that no deleterious effects on calcium metabolism of this particular brand of cocoa in the amounts consumed were revealed. For all periods and subjects, the calcium balances on the cocoa diets averaged — 9.74 mg. per day and on the noncocoa diets — 12.37 mg. per day, the difference being insignificant statistically. The calcium intakes on the cocoa and noncocoa diets averaged very nearly the same, 604 and 590 mg. per subject daily respectively. Among twenty-seven comparisons of calcium balances involving each subject on each diet, with and without cocoa, sixteen favored the cocoa diet and eleven the noncocoa. The deviation of 2.5 from the ideal outcome of 13.5 if random factors alone operated is somewhat less than the standard deviation of twenty-seven events, each of which, with equal probability, may result in either of two ways, i. e. $\sqrt{0.5 \times 0.5 \times 27} = 2.6$. Hence such a deviation may well have resulted from chance only. An application of the analysis of variance to each diet comparison confirmed the essentially negative significance of the data.

These negative results may mean that in human subjects the cocoa used has no effect on calcium utilization. On the other hand, they may mean that the method of study is not sufficiently sensitive to detect an effect of cocoa because of the variation among the data within diet periods and within subjects due to uncontrolled factors. A statistical analysis of this variation showed that for all four diet periods the variation was such that average differences of 116, 57, 145 and 25 mg. respectively in calcium balance per subject daily induced by a dietary factor such as cocoa would have been detected with a probability of 0.05. This means that the uncontrolled intraperiod and intragroup variation would be expected to produce an average difference in calcium balance of these magnitudes (or greater) only once in twenty trials. With the final low calcium diet, which was unsupplemented with cocoa for twelve days for each subject and supplemented with 21 Gm. of cocoa daily for an adjacent twelve day period, the uncontrolled variation was so much less than with that of the other diets that an average difference in calcium balance of only 25 mg. could have been detected with a probability of 0.05. Twenty-one Gm. of cocoa contains 105 mg. of oxalic acid, capable of combining with 46 mg. of calcium. On the other hand, 21 Gm. of cocoa itself contains 30 mg. of calcium, enough to combine with two thirds of the oxalic acid contained in it. In another diet period in which the critical difference in calcium balance detectable at the 5 per cent probability level was 57 mg., the daily cocoa supplement amounted to 1 ounce (28.35 Gm.) and contained 142 mg. of oxalic acid, sufficient to combine with 63 mg. of calcium. In this case the cocoa supplement contained 41 mg. of calcium, leaving an excess of oxalic acid sufficient to combine with only 22 mg. of calcium contained in the basal foods. These calculations illustrate the difficulty of detecting the effect of cocoa on calcium metabolism in the human being if the effect is due entirely to the oxalic acid contained in it.

Any discussion of the possible effect of cocoa on the utilization of dietary calcium in the human organism should consider the possibility that calcium oxalate is considerably more efficiently utilized in the human organism than in the body of the rat. The rat has been shown [10] to be almost completely incapable of utilizing calcium oxalate or the calcium of spinach, a vegetable containing more than enough oxalic acid to combine with its content of calcium. That the human body utilizes the calcium of spinach to a considerable extent is indicated clearly by the experiments of

6. Millat, L.: Toxicité des coques de cacao et influence de celle-ci sur la toxicité de la caféine, Compt. rend. Acad. d. sc. **213:** 591, 1941.

7. Neumann, R. O.: Die Bewertung des Kakaos als Nahrungs- und Genusmittel: Experimentelle Versuche am Menschen, Arch. Hyg. **58:** 1, 1906.

8. These experiments as well as the rat-feeding experiments were aided by funds granted to the University of Illinois by the National Dairy Council on behalf of the chocolate industry and the International Association of Ice Cream Manufacturers.

9. The amount of cocoa contained in 400 cc. of chocolate milk containing 1.4 per cent of cocoa. Incidentally this amount of cocoa, 5.5 Gm., approximates the average daily consumption in this country.

10. Fairbanks, B. W., and Mitchell, H. H.: The Availability of Calcium in Spinach, in Skim Milk and in Calcium Oxalate, J. Nutrition **16:** 79 (July) 1938.

McLaughlin [11] and of Fincke and Garrison.[12] The experiments of Bonner and others [13] on children failed to detect an adverse effect on calcium utilization of 100 Gm. daily portions of canned spinach or of an amount of oxalic acid, 700 mg., contained in such portions. It is true that the calcium intake in these experiments, ranging from 800 to 1,300 mg. daily, was liberal for these subjects, but the oxalic acid doses used were capable of combining with over 300 mg. of calcium. Even so, the fecal excretion of calcium, which presumably would include the calcium oxalate thus formed, was only inconsiderably increased by an average of 28 mg. daily in the spinach periods and of 42 mg. in the oxalic acid periods.

On other grounds also the effect on calcium retention of oxalic acid, or any other agent capable of immobilizing calcium in the alimentary canal, would be much greater in the rat than in the human being. The rat is able to utilize non-oxalate calcium to a much greater extent than is man, and hence the precipitation of a given weight of calcium in the intestine by oxalic acid has a much greater effect on the calcium economy of the body. In the Illinois experiments on rats the calcium intake was retained to the extent of 89 per cent on the average by the animals on noncocoa rations. Hence the immobilization of 100 mg. of dietary calcium by the oxalic acid of cocoa would be expected to depress calcium retention by 89 mg. However, in the experiments on women the sparing effect of dietary calcium on body calcium averaged about 19 per cent, in agreement with many other tests reported in the literature. In this case the immobilization of 100 mg. of dietary calcium by oxalic acid would reduce calcium retention by only 19 mg.

An interesting finding in the experiments on human subjects reported here in a preliminary way was that the tolerance limit for cocoa was about 1 ounce daily. Larger portions induced headache, nausea and impaired appetite. To consume the equivalent of 1 ounce of cocoa containing 20 per cent fat in the form of sweet chocolate prepared with the minimal proportion of chocolate liquor, 15 per cent,[14] one would have to consume approximately 12 ounces. A detailed report of both the rat and the human experiments will be published elsewhere.

CONCLUSIONS

In the growing rat, two medium cost cocoas containing about 22 per cent of fat depressed the utilization of calcium in proportion to their content of oxalic acid. A low cost cocoa containing about 11 per cent fat and apparently appreciably more cacao shell depressed growth more than the higher grade cocoas and depressed calcium retention about twice as much as can be accounted for by its content of oxalic acid.

In adult human subjects, whether subsisting on a low calcium diet or on diets of borderline calcium adequacy, a medium cost cocoa in daily portions of 21 to 38 Gm., approximating the tolerance limit, was not shown to affect the calcium balance adversely.

However, if the effect of such cocoas on calcium utilization resides entirely in their content of oxalic acid, as the rat experiments indicate, it would not be expected to be appreciable in human metabolism, because the oxalic acid content of cocoa is only moderate, i. e. 0.5 to 0.6 per cent, and the calcium in the cocoa itself is sufficient to combine with about two thirds of the oxalic acid. Furthermore, the immobilization of milk calcium in the digestive tract by the remaining third of the oxalic acid of cocoa, equivalent to 22 mg. of calcium for a 1 ounce portion of cocoa, would be expected to depress calcium retention by only one fifth of this amount (4 or 5 mg. of calcium), because of the poor utilization of calcium (19 per cent or so) by the adult human organism. Such a small depression in calcium retention is practically impossible to detect by the technic of the metabolism experiment with human subjects.

The effect of low cost, low fat (dry) cocoas, rather commonly used in this country at times, should be tested on human subjects with reference to tolerance and to calcium utilization, since with these cocoas other constituents than oxalic acid may depress the utilization of dietary calcium.

11. McLaughlin, L.: Utilization of the Calcium of Spinach, J. Biol. Chem. **74**: 455, 1927.
12. Fincke, M. L., and Garrison, E. A.: Utilization of Calcium of Spinach and Kale, Food Res. **3**: 575, 1938.
13. Bonner, Priscilla; Hummel, Frances C.; Bates, Mary F.; Horton, James; Hunscher, Helen A., and Macy, Icy G.: The Influence of a Daily Serving of Spinach or Its Equivalent in Oxalic Acid on the Mineral Utilization of Children, J. Pediat. **12**: 188 (Feb.) 1938.
14. U. S. Federal Register **9**: 14335 (Dec. 6) 1944.

ACCEPTED FOODS

The following additional foods have been accepted as conforming to the Rules of the Council on Foods and Nutrition of the American Medical Association for admission to Accepted Foods.

GEORGE K. ANDERSON, M.D., *Secretary.*

CANNED AND DRIED FRUITS AND FRUIT PRODUCTS (*See* Accepted Foods, 1939, p. 70).

Florida Citrus Canners Cooperative, Lake Wales, Fla.

DONALD DUCK, PRICELESS BRAND AND SEALDSWEET BRANDS CONCENTRATED ORANGE JUICE. The freshly squeezed juice is concentrated under a high vacuum and then filled hot into cans, which are sealed, flash pasteurized at 200 F. and then held in 40 F. storage.

Analysis (submitted by manufacturer).—Moisture 35.0%, total solids 65.0%, acid (as citric) 5.0%, invert sugar 25.0%, total sugar 50.0%, protein (N × 6.25) 4.3%, pectin 0.3%, volatile oil 0.0025%, ascorbic acid 215 mg., phosphorus 120 mg., potassium 1,600 mg., calcium 80 mg., magnesium 78 mg., manganese 0.14 mg., iron 0.1 mg., aluminum 0.3 mg. per hundred cubic centimeters, specific gravity 1.318.

Calories.—In the concentrate, 2½ per gram. In the diluted product, ½ per gram.

Vitamins.—Ascorbic acid (titration method) in juice reconstituted with six volumes of water contains not less than 40 mg. per hundred cubic centimeters.

PREPARATIONS USED IN THE FEEDING OF INFANTS (*See* Accepted Foods, 1939, p. 156)

Harold H. Clapp, Inc., Rochester, N. Y.

CLAPP'S JUNIOR FOODS PEARS, consisting of pears and sugar.

Analysis (submitted by manufacturer).—Total solids 16.27%, crude fiber 1.26%, protein (N × 6.25) 0.30%, ash, 0.23%, fat (ether extract) 0.05%, carbohydrate (by difference) 14.43%, phosphorus (P) 9.6 mg. per hundred grams, calcium (Ca) 8.7 mg. per hundred grams, iron (Fe) 0.7 mg. per hundred grams, copper (Cu) 0.11 mg. per hundred grams.

Calories.—16.7 per ounce; 0.59 per gram.

Vitamins.—Ascorbic acid 3.6 mg. per hundred grams, riboflavin 0.025 mg. per hundred grams.

Heinz Company, Pittsburgh.

HEINZ STRAINED APRICOTS WITH OATMEAL contains fresh apricots, oat flour, sugar and water.

Analysis (submitted by manufacturer).—Total solids 25.79%, total sugar as sucrose 19.11%, acidity as citric 0.42%, protein (N × 6.25) 0.90%, fat (by acid hydrolysis) 0.28%, crude fiber 0.61%, ash 0.37%, salt 0, total carbohydrates other than crude fiber by difference 23.21%, calcium 10.0 mg. per hundred grams, phosphorus 22.0 mg. per hundred grams, iron 0.5 mg. per hundred grams, copper 0.11 mg. per hundred grams, lead 0.06 mg. per hundred grams (0.0042 grains per pound), arsenic 0.

Calories.—0.99 per gram, 28 per ounce.

Vitamins.—	I. U./100 Gm.	Mg./100 Gm.
Carotene	2,100	
Thiamine	.....	0.039
Riboflavin	.....	0.017
Ascorbic acid	.....	3.45

THE JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION

535 North Dearborn Street - - Chicago 10, Ill.

Cable Address - - - - : "Medic, Chicago"

Subscription price - - - - - : Eight dollars per annum in advance

Please send in promptly notice of change of address, giving both old and new; always state whether the change is temporary or permanent. Such notice should mention all journals received from this office. Important information regarding contributions will be found on second advertising page following reading matter.

SATURDAY, NOVEMBER 24, 1945

THE NATIONAL HEALTH PROGRAM—THE PRESIDENT'S MESSAGE

In the Organization Section of this issue of The Journal appears the complete text of the message of President Harry S. Truman to the Congress, delivered on November 19. The text was received as The Journal was going to press. The President presents a five point program. The measures proposed by the Hill-Burton bill for increased funds for hospitals and health centers throughout the nation are covered by his first point. The American Medical Association has approved the principles of the Hill-Burton bill subject to safeguards which are in the text reported by the committee which conducted hearings on this measure.

The second recommendation of the President is for expanded maternal and child health services—essentially those proposed by the Pepper bill. It should be apparent that the passing of a nationwide compulsory sickness insurance bill ought to make unnecessary the kind of proposals included under the Pepper Maternal and Child Health measure.

The President urges increased funds for medical education to be given to public and nonprofit institutions for extending medical education and particularly for research in the fields of cancer and mental health. Obviously this proposal is duplicated to some extent by the proposals for the National Science Foundation. This proposal would place the federal government definitely in control of medical education throughout the United States through its ability to allocate funds to medical educational institutions.

The fourth proposal is for a nationwide system of compulsory sickness insurance to cover every man, woman and child in the United States and to care for the indigent through insurance policies purchased by local agencies for which they would be reimbursed in whole or in part by the federal government. The American Medical Association has opposed compulsory sickness insurance consistently for many years. The President reaffirms Senator Wagner's peculiar interpretation of the term socialized medicine by claiming that "this is not socialized medicine." The affirmation will not be convincing to the physicians of the United States who would be compelled to submit to politically controlled medicine should such a measure ever become the law of the nation.

Finally, the President urges compensation of workers for disability due to illness. The House of Delegates of the American Medical Association has approved such proposals in the past.

Fortunately the House of Delegates of the American Medical Association is scheduled for a session to be held in Chicago, December 2-6. The House of Delegates will no doubt at that time state officially the point of view of the American Medical Association on the President's proposals.

THE PELLAGRA PROBLEM

The discovery that a deficiency of niacin is directly related to the development of pellagra in man and blacktongue in dogs has not completely clarified the etiology of these conditions. For example, the relatively high incidence of pellagra in maize-eating populations is not entirely thus explained, since corn contains approximately the same amount of niacin as many other foods, such as eggs, milk and oats.[1] Also the early observations of Goldberger and his associates that various protein foods and, indeed, the amino acids tryptophan and cystine exert some beneficial effect in human pellagra indicates that factors in addition to niacin may be involved.

The results of recent experimental studies on the pellagra problem appear to be valuable in elucidating these two puzzling questions. Blacktongue, the canine counterpart of pellagra in man, cannot be produced in dogs by a niacin low synthetic diet adequate in all other respects unless corn grits is added.[2] The effect of corn grits can be nullified by the addition of niacin.[3] Even the rat, a species notoriously resistant to lack of niacin, will show inhibition of growth when corn grits is added to the ration.[4] The effect is nullified by niacin, as in the dogs, or by increasing the protein intake. The most recent contribution to the problem [5] demonstrates that the addition of the amino acid tryptophan alone

1. Teply, L. J.; Strong, F. M., and Elvehjem, C. A.: J. Nutrition **23**: 417 (April) 1942.
2. Handler, Philip: Proc. Soc. Exper. Biol. & Med. **52**: 263 (April) 1943.
3. Krehl, W. A.; Teply, L. J., and Elvehjem, C. A.: Proc. Soc. Exper. Biol. & Med. **58**: 334 (April) 1945.
4. Krehl, W. A.; Teply, L. J., and Elvehjem, C. A.: Science **101**: 283 (March 16) 1945.
5. Krehl, W. A.; Teply, L. J.; Sarma, P. S., and Elvehjem, C. A.: Science **101**: 489 (May 11) 1945.

will likewise counteract the effect of corn grits, thus confirming experimentally an observation made long ago in pellagrins by Goldberger.[6]

A published explanation of the apparent synergistic relationship between tryptophan (and protein) and niacin, on the one hand, and the antagonistic effect of corn, on the other, is not yet forthcoming. However, the suggestion [6] that the synthesis of niacin by the intestinal flora may be involved appears logical. According to this view the addition of corn grits may decrease the number of niacin synthesizing micro-organisms in the intestinal flora and thus lessen the amount of niacin available to supplement an inadequate niacin intake. The addition of tryptophan or protein, on the other hand, may favor an increase of the niacin synthesizing organisms and thus nullify the effect of corn. The fact that the proteins of corn are comparatively low in tryptophan may also be a related factor. Further studies on this important problem will be awaited with interest.

RECONVERSION OF THE 9-9-9 HOUSE OFFICER PROGRAM

The Procurement and Assignment Service has now announced its program for reconversion of the 9-9-9 program (see page 878, this issue). The Army and Navy have agreed to the plan. The present interns, most of whom will complete nine months of internship about April 1, 1946, will be permitted to continue the internship until July 1, 1946. Should hospitals be unable to accommodate these men plus the new interns who will commence their appointments about April 1, those who hold commissions and have completed their nine months of intern training will be ordered to active duty. New interns appointed April 1, 1946 will be allowed to continue until July 1, 1947, by which time most medical schools hope to be graduating classes annually every June.

Commissioned officers who are serving as junior or assistant residents will be called on completion of their eighteen months of hospital work about April 1, 1946 and (senior) residents completing twenty-seven months will also be called at that time. Exceptions will be made in the deferment of commissioned officers to a second or third nine months hospital appointment only rarely, in "cases in which their services are absolutely essential" and "the Procurement and Assignment Service and the Surgeons General are convinced that every effort to obtain the services of veterans, physically disqualified or otherwise ineligible men or of women has been made."

The primary purposes of this plan are (*a*) to begin moving toward a peacetime status of twelve months internships commencing each July, while still providing replacements for the Army and Navy and (*b*) to provide more residency positions for returning physician veterans who are and will be seeking such training in large numbers. Hospitals are urged to "begin at once to appoint veterans to every staff position so that, with rare exception, every military resident can be called to active duty not later than April 1, 1946." As soon as a veteran is adequately trained to replace a commissioned officer resident, that resident will be called to active duty even before April 1, 1946. Hospitals are again reminded that veterans do not count on their house officer quotas. These regulations should be studied with care by all concerned.

The foregoing represents the new policy of the Procurement and Assignment Service and the Surgeons General of the Army and Navy. Unfortunately, other existing Army regulations are in conflict with the commendable purpose and successful operation of this plan. The Army apparently proposes to continue the A. S. T. P. in medical schools if Congress allocates funds and to require medical schools to continue the accelerated program even though relatively few A. S. T. P. students will remain in school. Should the Army persist in this unreasonable program, the plans of several medical schools to provide special work for physician veterans next spring or summer cannot be consummated. Furthermore, medical school calendars will be disoriented with the house officer program outlined by the Procurement and Assignment Service and inordinate confusion will result. The Army cannot justify a continuation of acceleration in medical schools desiring to decelerate, and the schools should refuse to accede to the demand.

RESTLESS LEGS

Axel Ekbom [1] reports a "hitherto virtually unknown disease which, because of its prevalence and the torment it causes in severe cases, is of great practical importance." The symptoms seem to be purely subjective and consist of peculiar paresthesias, a feeling of weakness in the legs and a sensation of cold in the feet. The paresthesias are the most characteristic signs of the disease and are therefore important for the diagnosis. They are experienced over the inner aspect of the legs, sometimes in the thighs but rarely in the feet. The arms may be involved. The patients have difficulty in clearly defining this sensation and refer to it most frequently as "crawling." Characteristically, the sensation develops only when the legs are still, most often after the patient retires for the night. For relief the patients move their legs continually or get up and walk about. Their sleep may be disturbed night after night for years with periods of improvement and exacerbation. In its most severe form the disease causes

6. Goldberger, Joseph, and Tanner, W. F.: Pub. Health Rep. **37:** 462 (March 3) 1922; **39:** 87 (Jan. 18) 1924; **40:** 54 (Jan. 9) 1925.

1. Ekbom, K. A.: Restless Legs, Acta med. Scandinav., supplement 158, 1945.

great suffering. The disease or syndrome occurs in two main forms: one characterized entirely or mainly by paresthesia and termed by the author "asthenia crurum paraesthetica" and the other, in which pain predominates and which the author calls "asthenia crurum dolorosa." The author uses the term "restless legs" for both groups. The author encountered the condition 18 times among 4,259 persons, 2,411 men and 1,848 women, who visited the neurologic outpatient service of the Serafimer Hospital, Stockholm, during the year 1944. Altogether the author observed, between 1943 and 1945, 34 moderately severe and severe cases of asthenia crurum paraesthetica, 15 cases of the asthenia crurum dolorosa and 65 cases in which the symptoms were so mild that they did not require any treatment.

The paresthetic variety is an easily recognizable condition, probably closely related to acroparesthesia and nocturnal burning feet. The painful variety consists of mild to moderate aching localized in the same situations as the paresthesia. The condition is often worse when the patient is resting and forces him to move his legs. Generally speaking, continuous paresthesia points to an organic lesion of the nervous system, while intermittent paresthesia of the type described in this syndrome points to a functional disorder possibly of vasomotor nature. The author's experience with two vasodilating drugs (Doryl, Merck, carbaminoylcholine chloride and Priscol, Ciba, benzylimidazolin hydrochloride) lend further support to the vasomotor theory of the condition. However, the author admits that the part which the treatment played in the improvement in the long run is difficult to determine.

A description of a similar syndrome was given by Wittmaack in 1861. This author referred to a condition described by older physicians as "anxietas tibiarum." The only other reference is to be found in an article published by Allison [2] in 1943 in which he speaks of "leg jitters" as a common minor ailment which he had never seen described. According to Allison the patients experience "a curious, unlocalized, restlessness in one or both legs. It is not quite a pain but is distinctly unpleasant. Massage has no effect, but getting up and walking about may give relief. The disagreeable sensation stops at once on chewing 1/100 grain of nitroglycerin, suggesting that the cause is vascular."

Ekbom believes that the reason why this syndrome of anxietas tibiarum has been forgotten is that the interest of the neurologists more recently has been centered on neuroses, the psychologic background receiving main attention. Little interest was paid to the clinical features of paresthesias during the twentieth century. The tendency is to label conditions with unusual symptoms as "hysteria," "psychoneurosis," "psychic pain" and the like.

2. Allison, F. G.: Obscure Pains in the Chest, Back or Limbs, Canad. M. A. J. **48**: 36 (Jan.) 1943.

Current Comment

APTITUDE TESTS FOR VOCATIONAL GUIDANCE

Tests for the appraisal of aptitudes and interests of high school students, with special reference to education and choice of a vocation, have achieved wide recognition. Certainly much useful information can be derived from the competent use of psychologic tests and of interest schedules. Tests of this kind have been used extensively by the military forces. There is good scientific evidence favoring the use of tests, but they must be given by people who know their interpretation and limitations. Unfortunately there are many promoters who advertise counseling services together with tests who do not themselves have technical competence in their use. These are probably the very men who come to the attention of the public more than those who are really qualified to give and interpret the tests properly. In differentiating between those who are competent it is useful to ask the qualifications of the senior man or owner of a firm that advertises such services. If he is himself a member of the American Psychological Association and if his examiners are also so qualified, one can assume that the services are reasonably satisfactory. A useful procedure is to make inquiry with the department of psychology in the nearest college. Such inquiry will usually bring information about the capable people in the locality. The value of vocational guidance is unquestionable but is definitely limited by the training of those performing the tests. Furthermore, aptitude testing cannot be considered infallible and must be viewed as subject to modification as the result of continued experience.

CHEMOTOXIC MENTAL DISEASE

Interest in mental diseases has increased because of new problems associated with the war. Mental patients occupy a larger number of beds in institutions than any other group of special patients, and for a longer period. Mental disease may be directly or indirectly related to infection (syphilis, encephalitis, malaria), endocrine disorders (pregnancy, thyrotoxicosis), cerebral arteriosclerosis and to various toxic chemicals. Undoubtedly the importance of chemotoxic factors is increasing on account of industrial developments, medicinal trends and habits associated with the war effort. The proper recognition of causal relations between neurotoxic chemicals and mental disorders becomes, therefore, more and more important for their control. Mental diseases may follow the chronic use of morphine, cocaine, marihuana, bromides, alcohol and occasionally barbiturates. Poisoning with nitrous oxide, potassium thiocyanide,[1] atabrine and sulfonamides [2] may also lead to

1. Solomon, J.; Greenblatt, M., and Coon, G. P.: Toxic Symptoms and Death Associated with Potassium Thiocyanate Therapy, New England J. Med. **229**: 241 (Aug. 5) 1943.
2. Little, S. C.: Nervous and Mental Effects of the Sulfonamides, J. A. M. A. **119**: 467 (June 6) 1942.

serious and sometimes prolonged psychotic episodes. Generally less appreciated is the psychotic action of several chemicals extensively produced and used in industry and in part sold to the public as constituents of finished products. Carbon monoxide,[3] carbon disulfide,[4] manganese, nicotine, lead and lead compounds, especially the volatile aliphatic compounds, mercurials, particularly the volatile alkyl derivatives,[5] and organic nitrites are examples. These chemicals have caused many accidental or occupational poisonings associated with transitory or permanent mental disorders. The action of some of these substances on the nervous system is insidious. Thus diethyl mercury and dimethyl mercury salts, which are used as fungicidal dressings for seed grain, may produce nervous symptoms only some weeks or months after exposure without causing at the same time manifestations of typical mercury poisoning. A similar specific neurotropic action is displayed by tetraethyl lead, which produces cerebral symptoms because of its fat soluble properties. Close or prolonged contact with these agents may lead to permanent and progressive psychotic conditions.

THE TIME THAT PHYSICIANS SPEND WITH PATIENTS

Several efforts have been made to estimate the number of physicians necessary to supply satisfactorily medical care to a community by determining the amount of time required to care for patients. A recent report [1] states the time spent on their patients by six physicians engaged in private practice in a war industry area in 1944. The six physicians held office hours on a rotating schedule in two war housing projects. They held 318 office periods, comprising 1,602 patient visits, during the first three months of 1944. The average amount of time spent per patient by the six physicians varied widely—from 12.2 to 21.3 minutes; as more patients were seen during a specific office period the amount of time per patient decreased, thus representing a speed-up in the work of the physician. This suggested to the investigator that the physician is generally "aware of the number of patients waiting to see him and tended to adjust the speed of his work accordingly." The average time spent by six physicians in two war housing projects in seeing their patients cannot possibly give a true index of the adequacy of the medical care or the number of physicians which are necessary to care for the needs of any other population group. Patients differ enormously in the amount of care needed, not only between patient and patient, but between the same patient on different occasions. Different physicians also do not work with the same speed or efficiency. Indeed, there are so many factors involved—nature of disease, talkativeness of patient or doctor, procedures attempted in single visit, nature of the communities, conditions of practice and the like—that these averages are quite meaningless. The only criterion which can satisfactorily meet the ideals of the medical profession involves the expenditure of as much time by the physician for each patient as is necessary to make a correct diagnosis and to institute the best available therapy on that patient. Even though this ideal is not always attained, even in the practices of physicians in housing projects, physicians generally cannot accept any yardstick that might interfere with this objective.

3. Sanger, E. B., and Gilliland, W. L.: Severe Carbon Monoxide Poisoning with Prolonged Coma, J. A. M. A. **114:** 324 (Jan. 27) 1940.
4. Braceland, F. J.: Mental Symptoms Following Carbon Disulphide Absorption and Intoxication, Ann. Int. Med. **16:** 246 (Feb.) 1942.
5. Koelsch, F.: Gesundheitsschädigungen durch organische Quecksilberverbindungen, Arch. f. Gewerbepath. u. Gewerbehyg. **8:** 113, 1937. Hill, W. H.: A Report of Two Deaths from Exposure to the Fumes of di-Ethyl Mercury, Canad. J. Pub. Health **34:** 158 (April) 1943. Hunter, D.; Bomford, R. R., and Russell, D. S.: Poisoning by Methyl Mercury Compounds, Quart. J. Med. **9:** 193 (July) 1940.
1. Davis, Burnet M.: A Note on Physician Time Per Patient in Private Practice, Pub. Health Rep. **60:** 1113 (Sept. 21) 1945.

SHORTAGE OF PENICILLIN

Apparently a definite shortage of penicillin for injection prevails in many places, and physicians are asking the reason for this shortage. A number of factors have produced the present short supply. Included are strikes, difficulty in maintaining production because of contamination during fermentation, increased export, the use of poor quality corn steep liquor and the manufacture of penicillin mixtures. Steps are being taken to overcome the penicillin shortage. Export controls have been suggested to permit records of the quantity of penicillin leaving the United States. Directives giving hospitals first call on penicillin production have also been proposed. A higher grade of corn steep liquor is being sought. Another step which should be undertaken by the manufacturers until the present shortage is alleviated is the discontinuance of doubtfully acting mixtures. Penicillin for injection purposes is truly a life-saving measure and its distribution should be given priority over the manufacture and distribution of over-the-counter items of less value. Judging from letters and telegrams that have come to the headquarters of the American Medical Association, resentment is developing among physicians unable to secure this product for serious cases of disease.

YEAST AS FOOD

Attention has been called [1] to the manifold ways in which micro-organisms have been useful to man. Such processes as the curing of cheese, leavening of breads and fermentation of wines employ the peculiar metabolic activity of the yeast, mold or bacteria concerned; during the recent war the practicability of using the micro-organism itself as a food has been demonstrated, notably with yeast. Postwar needs for nutriment have led the British government to stimulate the production of yeast in Jamaica and in other islands of its British West Indies where sugar cane can be grown.[2] The yeast isolated from sugar cane is not only a rich source of protein of high biologic value but also contains all of the B vitamins and, if present plans mature, should provide these dietary essentials at relatively low cost. By thus utilizing the natural resources of these islands, progress may be made in combating the poverty so long associated with this region.

1. Syntheses in the Intestine, Current Comment, J. A. M. A. **126:** 174 (Sept. 16) 1944.
2. Chem. & Engin. News, **23:** 1656 (Sept. 25) 1945.

MEDICINE AND THE WAR

PROCUREMENT AND ASSIGNMENT SERVICE FOR PHYSICIANS, DENTISTS AND VETERINARIANS

RECONVERSION OF THE 9-9-9 PROGRAM

The 9-9-9 program was devised in order to supply American hospitals with residents. Without it every commissioned officer, which represented 80 per cent of each class, would have been called to active duty at the end of twelve months of internship and the American hospitals would have been practically devoid of residents. The Procurement and Assignment Service made arrangements with the Surgeons General so that a sufficient number of officers could have their active duty orders delayed and the hospitals could have a minimum supply of residents with which to operate during the war. The whole plan constituted a loan of military personnel to civilian institutions by the Surgeons General, and they were willing to enter into the plan because it was perfectly obvious that there was no other supply available with which to keep these hospitals operating.

Increasing numbers of veterans who have seen active duty are becoming available for filling such positions, and therefore the Surgeons General are convinced that it is in order to call to active duty as rapidly as possible every commissioned officer who has not been on active duty. Hospitals, therefore, must begin at once to appoint veterans to every staff position so that, with rare exception, every military resident can be called to active duty not later than April 1, 1946. As soon as a veteran has been adequately trained to replace a commissioned officer resident, that officer should be called to active duty at once without regard to whether or not he has completed the full term of his present deferment. The hospital must notify the state chairman for physicians of the Procurement and Assignment Service of the availability of such officers at once. State chairmen must watch these developments closely. At present veterans will not count in hospital quotas and it will be of great advantage to the hospitals to accept veterans as replacements for the officers who do count in quotas. The great advantage to hospitals in accepting veterans promptly is that they will be orientated to their position before April 1 in anticipation of the fact that after July 1 such veterans will be practically the sole source of supply of residents for civilian hospitals.

Hospitals and commissioned officers now serving as residents are reminded that any contract with hospitals is wholly subsidiary to their legal obligation to the Army and Navy and that each individual is subject to active duty orders at the discretion of the Surgeons General.

Present deferments were granted by the Surgeons General prior to VE day when there was no other supply available to the hospitals. In many institutions veterans are applying by the scores for hospital appointments, and hospitals should accept them to the greatest extent possible in anticipation of the fact that those not having been on active duty will be called.

In those cases in which the hospital desires the services of an officer on active duty in the Army to fill a residency and the hospital is willing to release a deferred commissioned resident who has not been on active duty before the termination of his present deferment period in exchange and he is an officer (1) who has been on active duty for two years or more, (2) who is now stationed within the continental United States and (3) who would accept the appointment, a request for the release of that officer under War Department Circular 296 through the Procurement and Assignment Service will receive immediately prompt and favorable consideration by the Surgeon General of the Army. This replacement possibility does not extend to officers on duty with the Navy.

The Procurement and Assignment Service will continue to recognize its responsibility toward the hospitals, and in those individual instances, very limited in number, in which hospitals are unable to secure a veteran after exhausting every effort or an officer who qualified under the War Department Circular 296 will be given consideration by the Procurement and Assignment Service and the Offices of the Surgeons General. Subject to the foregoing general provisions the reconversion from the 9-9-9 program to a peacetime twelve month service will be conducted in accordance with the following plan: (This outline applies to the April group. Information concerning those who finish their present term before April and after April will be released in the near future.)

SECTION I

A. Each commissioned officer who is an intern terminating his nine months of internship on April 1, 1946 will continue in his present internship until July 1, 1946. (For exceptions see section II.)

B. Each commissioned officer who is a junior resident terminating his junior residency on April 1 (if he has not already been replaced by a veteran prior to April 1) will be called to active duty on April 1 except in rare cases in which special request for his service is submitted to and approved by the Procurement and Assignment Service and the Surgeon General, such request to be effective only until he is replaced by a veteran and not beyond July 1, 1946.

C. Each commissioned officer who is senior resident and who is completing his twenty-seven months of service in a hospital following graduation will be called to active duty on or about April 1, 1946 if not replaced prior to that date by a veteran.

D. Senior students who graduate on or about April 1, 1946 are to be accepted for internship on that date. This new group of interns will be allowed to serve internships until July 1, 1947. This means that hospitals would have a maximum of twice their quota of interns from April to July and their quota of interns from July to the following July. In view of this and in order to maintain equitable distribution of the intern supply, quotas for interns will be maintained at their present levels.

SECTION II

A. The present interns (referred to in paragraph A, section I) will be called to active duty on July 1, 1946 except (1) those who are called to duty between April 1 and July 1 because the hospitals cannot facilitate both the old and new groups of interns for three full months of overlapping service; and (2) those rare cases in which their services are absolutely essential for an additional period in civilian hospitals. Their services will be deemed necessary only in those cases in which the Procurement and Assignment Service and the Surgeons General are convinced that every effort to obtain the services of veterans, physically disqualified or otherwise ineligible men or of women has been made. [N. B.: If Congress votes funds to continue the ASTP beyond June 30, 1946 the military group in the following class (unless the Army Training Service abandons its present accelleration demands) will become available for internship on Jan. 1, 1947. This would produce a six month overlap from January to July, but it is hoped that this can be avoided.]

B. The present junior residents (referred to in paragraph D, section I) will be called to active duty as soon as replaced by a veteran with the same exceptions stated in paragraph A, section I.

C. (Blank).

D. The new graduates (referred to in paragraph D, section I), who will complete twelve months of internship on or before July 1, 1947 will be called to active duty on that date. These men may have as much as fifteen months of internship, that is, April 1, 1946 to July 1, 1947.

SECTION III

By April 1, 1946 hospitals will be almost entirely dependent on nonmilitary personnel to fill residencies.

This program does not contemplate an interference with the present policy of not including veterans in quotas. In fact, it emphasizes the importance of acquiring the services of such veterans at the earliest possible moment so that they will be adequately trained to serve as replacements for officers now deferred to April 1 and for the period when they will represent practically the entire supply—after July 1, 1946.

In the interest of adequate distribution of the available supply of veterans, it is expected that hospitals will not fill their house staff out of proportion to present quotas. These veterans should be used first as replacements, and present quotas for residents should not be exceeded until all deferred commissioned officers who have not been on active duty have been released from that particular hospital. This is not contrary to the policy of not counting veterans in quotas until they are sufficiently trained to be used as replacements.

It will not be necessary to submit forms 218 for the continuation of interns for the period from April 1 to July 1, referred to in paragraphs A and B, section I. It will be presumed, however, that the junior residents referred to in paragraph B, section I, practically without exception, will not be requested for this period and will be released prior to but not later than April 1 and as soon as they can be satisfactorily replaced by officers discharged or separated from the service.

The necessity of reconverting from the 9-9-9 program as rapidly as possible cannot be emphasized too strongly and to the greatest extent possible for the purpose of returning to the July to July twelve month internship this coming year. The most available and safest supply of physicians to serve residencies will be these who have completed their military obligation to the nation during the war and the duration of the emergency.

ARMY

GENERAL KIRK RECEIVES DISTINGUISHED SERVICE MEDAL

Major Gen. Norman T. Kirk, Surgeon General of the Army, was recently awarded the Distinguished Service Medal by Gen. Brehon Somervell, commanding general of the Army Service Forces, in recognition of his "outstanding leadership . . . in directing the largest Medical Department in the history of the United States Army." According to the official citation, "By careful planning, efficient administration and dynamic example he [General Kirk] made possible extraordinary care for sick and wounded American soldiers—care which has never been equaled in any war. He supervised the selection of qualified medical specialists who were assigned to posts stretching from the forward areas to general hospitals in the zone of the interior and combined their exceptional talents with a prompt evacuation system to achieve unprecedented results. Under his able guidance every effort was made to utilize the newer methods of medical and surgical treatment and to develop new technics. His plans for the care of battle casualties and outstanding efforts to prevent disease among the troops resulted in an enormous reduction in mortality and morbidity. Bearing tremendous responsibilities, General Kirk accomplished his trying task in a distinctly superior manner, thereby making a major contribution to the success of American arms." General Kirk graduated from the University of Maryland School of Medicine and College of Physicians and Surgeons, Baltimore, in 1910 and entered the service in 1912.

PLASTIC EYE CENTERS

In keeping with the Army's program to establish sufficient plastic eye centers to supply the needs of patients requiring prostheses, Fitzsimons and O'Reilly general hospitals, Denver and Springfield, Mo., respectively, have been added to the list of general hospital centers. Other army general hospitals now producing plastic eyes are Thayer, Birmingham, McGuire, Hammond, Barnes, Dibble, Newton D. Baker, Tilton (also Waltham Regional Hospital), Rhoads, Halloran, McCloskey, Wakeman, Glennan, Cushing, Ashburn, Crile, Northington, Gardiner, Lawson, Walter Reed, Percy Jones, Brooke, William Beaumont, Finney, Harmon and Valley Forge.

AWARD OF MERITORIOUS SERVICE UNIT PLAQUE

A Meritorious Service Unit Plaque was recently awarded to the 318th Medical Battalion for superior performance and outstanding devotion to duty from Nov. 15, 1944 to Sept. 20, 1945. This unit was charged with the important and difficult task of providing medical care, treatment and evacuation facilities for the entire personnel of the 93d Infantry Division, although they were distributed on various bases over a vast area of the Pacific. As the result of devotion to duty, long hours and cheerful cooperation on the part of all personnel, excellent medical care and treatment was provided in spite of the battalion being under strength in personnel, both commissioned and enlisted. Despite the difficult and trying situations the unit maintained high standards of administration and continued improvements in medical service, housekeeping, sanitation, military courtesy and discipline. The appearance of personnel was at all times exemplary. The unit average on ordnance inspections was among the highest in the 93d Infantry Division, notwithstanding the fact that class 2 and 3 vehicles comprised 90 per cent of the vehicles. The unit was at all times preeminent in its achievements and received commendations from various visiting officials, including the Surgeon General of the Army. Throughout the entire period of operation a high degree of morale and *esprit de corps* was maintained.

ARMY AWARDS AND COMMENDATIONS

Colonel Perrin A. Long

Col. Perrin A. Long, Baltimore, was recently awarded the Legion of Merit in recognition of his work as medical consultant to the surgeon, Mediterranean Theater of Operations, from Jan. 3, 1943 to Aug. 1, 1945. The official citation stated in part that, "as medical consultant to the surgeon, North African and later the Mediterranean Theater of Operations, Colonel Long efficiently organized the medical service of hospitals and other medical units and by his teaching and counsel developed such successful therapeutic methods of handling malaria, bacillary dysentery and other infectious diseases that mortality and permanent disability among American troops were reduced to an unprecedented low level." Dr. Long graduated from the University of Michigan Medical School, Ann Arbor, in 1924 and entered the service Aug. 27, 1942.

Colonel Arthur B. Welsh

The Legion of Merit was recently awarded to Col. Arthur B. Welsh, Washington, D. C. The citation stated that "he rendered conspicuous and meritorious service in the medical program at the Army from November 1941 to May 1945 in his capacity as deputy director, then as director of the Plans Division and later as deputy chief of the Operations Service, Surgeon General's Office. He envisioned, and established as his objective an all inclusive and comprehensive policy for the early treatment, hospitalization and evacuation of our sick and wounded soldiers. He formulated the Medical Department Mobilization Plan, which resulted in a well balanced medical service, as evidenced by the extremely low mortality rates during the all out prosecution of the war. He also formulated and effected the plan to increase the bed capacity of general hospitals employed overseas, resulting in an economy in the use of personnel and equipment at a time when the shortage of both was critical and the requirements greatest. His keen judgment and breadth of vision were apparent in the effectiveness of the operations of the Medical Service overseas." Dr. Welsh graduated from the University of Pittsburgh School of Medicine in 1925 and entered the service June 10, 1925.

Colonel Frank E. Stinchfield

The Legion of Merit was recently awarded to Col. Frank E. Stinchfield, formerly of New York, for "exceptionally meritorious conduct in the performance of outstanding service as commanding officer, ——— Station Hospital, during the period August 1943 to August 1944. Under adverse conditions Lieutenant Colonel Stinchfield developed this 4,000 bed capacity. In the short time he has operated this hospital it has rehabilitated and returned to duty approximately 85 per cent of its patients. Through his diligence, vision and ingenuity he developed a rehabilitation program so wide in scope that thousands of men were sent back to duty completely rehabilitated. The resultant conservation of manpower has materially aided the progress of the war. Lieutenant Colonel Stinchfield established two schools to make his extensive program of rehabilitation familiar to as many as possible. Faced with lack of proper equipment and a shortage of qualified personnel, he improvised his equipment and trained his own personnel. Since the inception of the program Lieutenant Colonel Stinchfield's staff has trained hundreds of officers in the principles and methods of rehabilitation." Dr. Stinchfield graduated from Northwestern University Medical School, Chicago, in 1935 and entered the service Feb. 15, 1942.

MISCELLANEOUS

WARTIME GRADUATE MEDICAL MEETINGS

The following subjects and speakers for Wartime Graduate Medical Meetings have just been announced:

California

Torney General Hospital, Palm Springs: The Rh Factor, Capt. George Macer, December 4; Hemolytic Streptococcus Respiratory Infections and Their Sequelae, Dr. Robert E. Solley, December 18.

A. A. F. Regional and Convalescent Hospital, Santa Ana Army Air Base: Contagious Diseases, Comdr. R. A. Trombley, December 4; Compound Fractures, Comdr. P. E. McMasters, December 18.

Camp Cooke Station Hospital, Lompoc: The Cancer Problem in Service Personnel, Lieut. J. S. Binkley, December 5; Problems in Tuberculosis, Comdr. W. L. Rogers and Comdr. A. W. Hobby, December 19.

Hoff General Hospital, Santa Barbara: The Cancer Problem in Service Personnel, Lieut. J. S. Binkley, December 5; Problems in Tuberculosis, Comdr. W. L. Rogers and Comdr. A. W. Hobby, December 19.

U. S. Naval Hospital, San Diego: The Classification and Diagnosis of the Anemias, Dr. A. G. Foord, December 6.

U. S. Naval Air Training Station, San Diego: Burns, Capt. H. T. D. Kirkbaum, December 7; The Penicillin Treatment of Syphilis and Gonorrhea, Comdr. W. W. Duemling, December 21.

U. S. Regional Hospital, Pasadena: The Rh Factor, Capt., George Macer, December 10.

Station Hospital, Fort Ord: Diseases of the Lungs and Their Treatment, Dr. Philip H. Pierson, December 15.

U. S. Naval Hospital, Treasure Island: Diagnosis of Atypical Anemias, Dr. Stacy R. Mettier, December 7.

Station Hospital, Camp Roberts: Diseases of the Thyroid: Clinical Considerations, Dr. Mayo H. Soley, December 8.

Station Hospital, Chico Army Air Base, Chico: Laboratory Aids in the Diagnosis of Disease, Dr. Jesse L. Carr, December 6.

Station Hospital, Stockton Air Field, Stockton: The Fundamentals of Endocrine Diagnosis, Dr. Roberto F. Escamilla, December 12.

U. S. Naval Hospital, Santa Margarita Ranch: Communicable Diseases, Major Norman Nixon, December 13; Problems Associated with the Surgery of the Biliary Tract, Capt., Howard K. Gray, December 27.

Dibble General Hospital, Menlo Park: The Surgical Anatomy of Hernia, Dr. Harold H. Lindner, December 3.

Illinois

Gardiner General Hospital, Chicago: Surgical Management of Tuberculosis, Dr. Jerome Head, November 28.

Pennsylvania

U. S. Naval Hospital, Philadelphia: Emotional Factors in Physical Illness, Dr. Earl D. Bond. December 7; Difficulties in the Diagnosis of Surgical Lesions of the Upper Urinary Tract, Dr. Leon Herman, December 28.

Virginia

Regional Hospital, Camp Lee, Petersburg: The Treatment of Vascular Hypertension by Extensive Sympathectomy, Dr. C. C. Coleman, December 14.

UNRRA NEWS

Plastic Surgery Team to Yugoslavia

According to a recent release from the United Nations Relief and Rehabilitation Administration, Washington, D. C., a plastic surgery team with special training and experience gained in England during the war will be dispatched to Belgrade, Yugoslavia, to train doctors and nurses there in the latest technics. Dr. Neville M. Goodman, director at London of UNRRA's European division of health, stated that "casualties among both soldiers and civilians in Yugoslavia created a large number of cases needing plastic surgery. The team of experts being sent by UNRRA will train enough Yugoslav surgeons so that they can carry on the necessary work without further assistance."

The team, which includes three surgeons, three specially trained nurses, a dental operator and a medical secretary, will be set up in a Belgrade hospital which escaped war damage and which is to be provided by the Yugoslav government. All the members of the team are drawn from the staff of a widely known British plastic surgery hospital near Basingstock. The team as a whole will remain in Yugoslavia for six months, but members will be relieved at intervals by different surgeons who will demonstrate and give training in the technics of other schools of plastic surgery.

CAPTAIN LOWELL T. COGGESHALL AWARDED GORGAS MEDAL

The Gorgas Medal, which has been awarded annually since 1942 by Wyeth Inc., Philadelphia, was given this year to Capt. Lowell T. Coggeshall (MC), U.S.N.R., expert on tropical diseases, "for distinguished service to our military forces in establishing new principles in the management of patients suffering from psychic disturbances as well as physical deterioration from the effects of malaria and filariasis. Captain Coggeshall instituted the system of psychosomatic rehabilitation by which thousands who faced discharge attributed to malaria and filariasis were returned to full duty. His was not only a brilliant accomplishment in a special field of military medicine, but he also inaugurated a new epoch in the treatment of a wide variety of diseases."

The medal was presented to Captain Coggeshall by Frank F. Law, vice president of Wyeth Inc., at a dinner in his honor at the Mayflower Hotel, Washington, D. C., October 29 and carries with it an honorarium of $500. The award was established in memory of Surg. Gen. William Crawford Gorgas, U. S. Army.

DALLAS COUNTY MEDICAL SOCIETY LISTS RETURNED MEMBERS

A campaign is being sponsored by the Dallas County Medical Society and the Dallas Southern Clinical Society whereby its members who have returned from service will be listed in each of the two Sunday papers published in that city. A different group of names is used each month and the campaign is to be continued for a minimum of six months.

In addition, a placard with the following legend is displayed in the reception room of every Dallas physician:

"May We Suggest, WHEN YOUR DOCTOR RETURNS FROM THE SERVICE RETURN TO YOUR DOCTOR."

PHYSICIANS SEPARATED FROM SERVICE

ARMY MEDICAL CORPS OFFICERS RECOMMENDED FOR/OR RELIEVED FROM ACTIVE DUTY

Alabama

Ferguson, Hal, 1st Lt., T. C. I. Hosp., Fairfield.
Goldsmith, Edward F. Jr., Capt., 22 Clinton Ave., Toulminville.
Gully, Virgil S., Capt., Butler.
Parker, Charles E., Major, 625 Woodley Rd., Montgomery.
Watkins, James H., Lt. Col., 1512 S. Hull St., Montgomery.
Williams, Howard B., Capt., 1514 Milner Crescent, Birmingham.

Arizona

Brainard, Hollis H., Capt., 2521 E. 8th St., Tucson.
Holbrook, William P., Col., Box 1390, Tucson.
Jolley, Elvie T., Capt., P. O. Box 776, Jerome.
Knight, Frederick W., Capt., Safford.
Littlefield, Jesse B. Sr., Lt. Col., 10 Calle Corta, Tucson.
Yount, Clarence E., Major, Prescott.

Arkansas

Crow, Merl T., Major, E. Cedar St., Warren.
Dozier, Floyd S., Major, Marvell.
Hogaboom, G. M., Capt., 914 Malvern, Hot Springs Nat'l Pk.
Huskins, James D., Capt., Siloam Springs.
Hyatt, Robert F., Capt., Monticello.
Kahn, Alfred Jr., Capt., 3504 Hill Rd., Little Rock.
Maynard, Ross E., Capt., 1216 W. 13th Ave., Pine Bluff.
Mossman, Frank D., Capt., 1922 Arch St., Little Rock.
Mundt, Leslie K., Lt. Col., 727 Franklin St., Helena.
Pierce, James O., Capt., 286 Home St., Marked Tree.
Rowland, Ely D., Major, 233 Hobson Ave., Hot Springs.
Sheppard, Jack M., Capt., 403 Church St., Eldorado.
Thompson, John K., Capt., 2601 S. Greenwood, Fort Smith.
Utley, Francis E., Major, Walls Hosp., Blytheville.
Whitehead, Robert H. Jr., Capt., 103 S. Harrison, Camden.

California

Andrews, A. V., Major, P. O. Box 365, Livingston.
Auerbach, Oscar, Major, 7215 Willoughby Ave., Los Angeles.
Axelrod, Bernard, Capt., 323½ S. Detroit St., Los Angeles.
Baker, John S., Major, 803 N. Detroit St., Los Angeles.
Barrett, Thomas F., Capt., 892 Ashbury St., San Francisco.
Bergman, George C., Lt. Col., 3514 W. 85th St., Inglewood.
Berkove, Alfred B., Lt. Col., 1 Hillwood Pl., Oakland.
Bernstein, Harry C., Major, 700 Parnassus Ave., San Francisco.
Bigotti, Richard T., Capt., 833 Pershing Ave., San Jose.
Coggin, Charles B., Major, 1537 Michigan Ave., Los Angeles.
Cruden, Alexander W., Capt., Sticoy Ventura Co.
Dittes, Albert G., Capt., 1712 E. Chevy Chase Dr., Glendale.
Dufficy, Rafael G. Jr., Capt., 324 Arguelo Blvd., San Francisco.
Fessler, Willard G., Capt., Los Altos.
Fogerty, Clement A., Col., Box 224, Ross.
Forcade, William P., Major, 254 Roman St., San Francisco.
Gompertz, John L., Major, 305 Hillside Ave., Piedmont.
Gottlieb, Jacob D., Major, Box X, Talmage.
Gummes, Glen H., Capt., 5721 Olympic Blvd., Los Angeles.
Hartwell, Brace F., 1st Lt., 196 6th St., Beaumont.
Havenhill, Asher D., Capt., End of Emeline St., Santa Cruz.
Hedge, Arden R., Major, 3040 Lyon St., San Francisco.
Hoff, Cecil W., Capt., 2797 Stoddard, San Bernardino.
Huff, William C., 1st Lt., Ventura Co. Hosp., Ventura.
Irvine, Wendell C., Major, 700 Roosevelt Bldg., Los Angeles.
Jackson, Edward A., Major, 236 Broadway, Atwater.
Kelley, Thomas J., Capt., 5335 Crenshaw Blvd., Los Angeles.
Kimball, Herbert S., 1st Lt., 19 Eastwood Ct., Orinda.
Knott, James I., Major, 3401 Pershing Dr., San Diego.
Laubersheimer, Anton, Capt., 829 S. Alvarado St., Los Angeles.
Linne, Francis B., Major, 1201 E. 8th St., Los Angeles.
Liston, Edward, Major, 1300 Hamilton Ave., Palo Alto.
McCarry, Frank, Capt., 4614 Sunset Blvd., Los Angeles.
Michels, Leon M., Major, 165 Stanyan St., San Francisco.
Michenner, John M., Major, 1398 Sheridan Ave., Pomona.
Moore, Carl L., Capt., 2300 Truxtun, Bakersfield.
Nicola, Quintus, Lt. Col., 112 S. Taylor Ave., Montebello.
Norquist, Donald M., Major, 640 W. 27th St., Los Angeles.
Pelton, Eugene C., Capt., 1400 Fell St., San Francisco.
Pettet, Charles H., Capt., 2200 W. 3d St., Los Angeles.
Prejean, Oran V., Major, 921 Louise St., Santa Ana.
Rombeau, Lee P., 1st Lt., 711 N. Dillon St., Los Angeles.
Sohler, F. E. Jr., Lt. Col., Corner West & 3d Sts., Gloverdale.
Taw, Richard L., Capt., 1137½ S. Doheny, Los Angeles.
Tuch, Benjamin E., Capt., 316 N. Rossmore, Los Angeles.
White, Marcus D., Col., Ontario.
Whitelock, Thomas S., Capt., 4778 Panorama Dr., San Diego.

Colorado

Ambler, John V., Lt. Col., 1925 Holly St., Denver.
Baker, Albert B., Lt. Col., 425 Cullembine St., Sterling.
Beuchat, Eugene S., Lt. Col., 602 E. 2d St., Trinidad.
Desmond, William M., Capt., Ordway.
Ley, Eugene B., Major, Pueblo.
Mihalick, John, Major, 611 E. Columbia, Colorado Springs.
Moffatt, Thomas W., Major, St. Joseph's Hos., Denver.
Morrill, Edgar M., Major, 1200 S. College Ave., Fort Collins.
Newland, Donald E., Capt., 510 Eudora St., Denver.
Swanson, Howard E., Major, 1589 Madison St., Denver.
Swiger, William B., Capt., 40 Eudora St., Denver.
Tramp, Paul E., Capt., Sheedy Bldg., Yuma.
Wills, Charles B., Capt., St. Lukes Hosp., Denver.

Connecticut

Albamonti, Mario J., Capt., 59 St. Regis Ave., Norwich.
Brown, Harwin J., Capt., 520 E. 8th St., Winfield.
Carpentieri, Anthony L., Capt., 727 Howard Ave., New Haven.
Castaldo, Louis F., Capt., 47 Goodsell St., Bridgeport.
Cipriano, Anthony P., Capt., 167 Livingston St., New Haven.
Cole, Milton J., Major, 110 Thomaston St., Hartford.
Dean, William W., Capt., New Haven Hosp., New Haven.
Dion, Asa J., Capt., 207 Washington St., Hartford.
Greenspun, David S., Capt., 153 Southwood R., Bridgeport.
Hansen, Paul S., Capt., Kings Corner, Essex.
Konopka, Frank J. Jr., Capt., 6 S. Whittlesey Ave., Wallingford.
Lemaitre, Paul G., Capt., 171 Providence St., Putnam.
Markle, Raymond D., Capt., Pleasant St., North Woodbury.
Patterson, Frederick A., Capt., 520 West Ave., Norwalk.
Tate, William J. Jr., Capt., 111 Kirtland St., Deep River.
Wesoly, Andrew S., Capt., 365 High St., New Britain.

Delaware

Baker, John H., Capt., 206 N. Walnut St., Milford.
Burton, Benjamin F. Jr., Capt., 214 S. State St., Dover.
Stradley, Shermer H. Jr., Capt., 2005 Monroe Pl., Wilmington.
Szatkowski, Eugene J., 1st Lt., 732 S. Broom St., Wilmington.

District of Columbia

Cliff, Arthur E., Major, 3901 S. Dakota Ave. N.E., Washington.
Dowd, James F., 1st Lt., 7815 12th St. N.W., Washington.
Farley, Otis R., Major, St. Elizabeth's Hosp., Washington.
Folston, Mortimer J., Capt., 506 22d N.W., Washington.
Gray, Luther W., Lt. Col., 128 18th St. S.E., Washington.
Hand, John P. Jr., Major, 3333 Baker St. N.E., Washington.
Kanof, N. B., Capt., 1312 Rittenhouse St. N.W., Washington.
Kay, Alvin I., Major, 3609 Van Ness St. N.W., Washington.
Kleinerman, Morris, Major, St. Elizabeths Hosp., Washington.
McKaig, Malcolm C., Major, 1473 Park Rd., Washington.
Maher, R. C. Jr., Capt., 315 W. Young St., Washington.
Moore, John S., Capt., 1868 Columbia Rd. N.W., Washington.
Settle, William B., Capt., 2630 Nichols Ave. S.E., Washington.
Turner, P. R., Capt., 213 Baltimore, Takoma Pk., Washington.

Florida

Angel, Norman S., Capt., 1814 S. W. 22 Terr., Miami.
Babers, Henry J. Jr., Major, 312 E. Union St., Gainesville.
Bippus, William E., Capt., 601 Guaranty Bldg., W. Palm Beach.
Bourkard, Ernest R., Capt., Jackson Memorial Hosp., Miami.
Chapman, Jules B., Major, 2151 Pearl St., Jacksonville.
Diehl, Earl H., Lt. Col., 608 Wilkie St., Dunedin.
Gifford, John P., Major, Vero Beach.
Hurt, Floyd K., Major, 3634 Boone Park Ave., Jacksonville.
Kelley, Oscar L., Major, 324 Valley Forge Rd., W. Palm Beach.
Kibler, John M., Major, 640 Woodward Dr., Lakeland.
Langley, Francis H., Lt. Col., 190 18th Ave., St. Petersburg.
Lefholz, Rothwell, Major, 1009 Huntington Bldg., Miami.
Massey, James L., Capt., 204 N. Madison St., Quincy.
Merritt, James W. Jr., Lt. Col., Jackson Mem. Hosp., Miami.
Norris, H. S., Capt., 50 Fuller Wood Dr., St. Augustine.
Orr, Louis M., Col., Delaney & Gatlin Ave., Orlando.
Pomerance, Joseph B., Major, 235 9th St., Miami Beach.
Russell, Ralph E., Capt., 1201 Lake Weir Ave., Ocala.
Saslaw, Milton S., Major, 1238 S. W. 8th St., Miami.
Seltzer, Morris B., Capt., 417 N. Oleander Ave., Daytona Beach.
Silverman, Harry Z., Capt., 760 Collins Ave., Miami Beach.
Sisler, Bruce H., Lt. Col., Miami Beach.
Sullivan, Benjamin H., Capt., 1010 W. Henry St., Tampa.
Tugwell, Wilton E., Major, 1504 W. Intendencia St., Pensacola.
Vinson, Clifford E., Capt., Jackson Memorial Hosp., Miami.

PHYSICIANS SEPARATED FROM SERVICE

Georgia

Adams, Everett H., Capt., Gen. Del., Rutledge.
Archer, George F., Capt., 21 Peachtree Memorial Dr., Atlanta.
Bailey, Milus K., Major, 1106 Med. Arts Bldg., Atlanta.
Bedingfield, Walter H., Capt., Rentz.
Brown, Samuel Y., 1st Lt., 1697 Noble Dr. N.E., Atlanta.
Chambers, James W., Major, 601 Winzor Ave., LaGrange.
Colvin, Emmett D., Major, 1259 Clifton Rd. N. E., Atlanta.
Cook, James T. Jr., Capt., Emory University Hosp., Emory.
Cook, Wendell H., Capt., Fort Oglethorpe.
Crawford, William B. Jr., Major, Gordonston, Savannah.
Danial, Abram B., Capt., Statesboro.
Fuller, William A. Jr., Major, 976 Piedmont Ave. N.E., Atlanta.
Garten, Leonard, Capt., University Hosp., Augusta.
Gower, Orien T. Jr., Lt. Col., 501 13th Ave. E., Cordele.
Griffin, Louie H., Capt., Warren A. Candler Hosp., Savannah.
Harrold, Charles C. Jr., Capt., 550 Orange St., Macon.
Henry, Charles G., Major, 2627 Helen St., Augusta.
Hicks, David Y. Jr., Major, Univ. Hosp., Augusta.
Hilsman, Palaemon L., Capt., 206 N. Jefferson St., Albany.
Jennings, James L., 1st Lt., 683 Elkmont Dr. N.E., Atlanta.
Johnson, Joseph E. Jr., Capt., 385 E. Church St., Elberton.
Jordan, Charles G., Major, Sasser.
Kandel, Harry M., Lt. Col., 432 Abercorn St., Savannah.
Leroy, Albert G., Capt., Tifton.
Levin, Harold B., Major, 116 Brown Pl. S.W., Atlanta.
McGehee, John M., Capt., 223 Jule Peek Ave., Cedartown.
Monfort, John M., Lt. Col., 3870 Club Dr. N.W., Atlanta.
Muecke, Harold W., Capt., 120 Hill Crest Ave., Macon.
Mulkey, Arnold P., Capt., 115 Winthrop Ave., Millen.
Neighbors, Joseph B. Jr., Major, 302 Hines Terrace, Macon.
Peck, Robert E., 1st Lt., 1246 Avon Ave. S.W., Atlanta.
Sanchez, Simeon E. Jr., 1st Lt., Barwick.
Storey, William E., Capt., Macon Rd., Columbus.
Warren, Joseph E., Capt., Hartwell.
Ward, Francis O., Major, Macon Hosp., Macon.
Worthy, William S., Capt., 26 Tanner St., Carrollton.
Yuckman, William, Major, Quitman.

Idaho

Bischoff, George H., Major, 6th & Bancock, Boise.
Douglas, Wallace S., Col., 1511 Prospect Ave., Lewiston.
Goodwin, Ralph A., Major, 115 N. Washington Ave., Emmett.
Jacobs, William R., Capt., 1204 12th, Lewiston.
Mellor, Wendell J., Capt., 492 L St., Idaho Falls.
Randall, Reginald C., Capt., Kellog.

Illinois

Alford, Frank L., Capt., 4 W. Crystal Lake Ave., Crystal Lake.
Augenlight, Jacob C., Capt., 1423 Farragut Ave., Chicago.
Axel, Carl, Capt., Lee.
Bailis, Jack M., Capt., Pittsfield.
Bard, Eli, Capt., 1938 Wilmot Ave., Chicago.
Behrents, Ellis G., Capt., Presbyterian Hosp., Chicago.
Bellows, John G., Major, 720 N. Michigan Ave., Chicago.
Block, Arnold S., 1st Lt., 1100 N. Dearborn St., Chicago.
Bloom, Charles R., Major, 1331 S. Austin Blvd., Cicero.
Boeke, Emmert G., Capt., Winslow.
Boon, Clifton I. U., Capt., 416 Ingleside Ave., Aurora.
Boshes, Benjamin, Lt. Col., 4918 Drexel Blvd., Chicago.
Brunner, Matthew J., Capt., 3518 W. Monroe St., Chicago.
Brownstein, Stanley, Capt., 5553 W. Congress St., Chicago.
Buehrig, Robert C., Major, Minier.
Busch, Robert C., Capt., 1340 Independence Rd., Chicago.
Cherner, Abraham M., Capt., 7641 S. Phillips Ave., Chicago.
Chobot, George R., Capt., 2515 S. Avers Ave., Chicago.
Czeisler, Tibor, 1st Lt., 770 S. Bennett Ave., Chicago.
Daro, August F., Major, 30 N. Michigan, Chicago.
Davenport, Harold A., Lt. Col., 303 E. Chicago Ave., Chicago.
Davis, Earl S., Capt., 421 E. Lincoln, Belvidere.
Deering, David, Capt., 999 Victoria St., Antioch.
Dickey, Marvin M., Lt. Col., Richmond.
Eisele, Owen J., Capt., 1761 N. 40th St., East St. Louis.
Ennis, Arthur L., Capt., 1349 W. Wood St., Decatur.
Faigle, Ernest T., Capt., 4310 N. Keystone Ave., Chicago.
Faller, Adolph, Lt. Col., 5206 S. Kimbark Ave., Chicago.
Fara, Frank J., Lt. Col., 5539 W. Cermak Rd., Cicero.
Flexman, Edmund A., Capt., 2534 Gunnison St., Chicago.
Friedman, David, Capt., 1532 3d St., Madison.
Gaynes, Harvey E., Lt. Col., 720 Waveland Ave., Chicago.
Giles, Roscoe C., Lt. Col., 541 E. 46th Pl., Chicago.
Goldstein, Hyman H., Major, 6500 Irving Blvd., Chicago.
Goldfine, Manuel, Capt., 841 E. 63d St., Chicago.
Gordon, Marion L., Capt., 1111 N. Monticello Ave., Chicago.

Illinois—Continued

Greene, B. L., Major, Elgin State Hosp., 750 S. State St., Elgin.
Harmon, Thomas F., Capt., 1601 S. Douglas St., Springfield.
Hipsking, Myron M., Major, 6060 Drexel Ave., Chicago.
Hoeffding, Waldemar, Capt., 102 E. Monroe St., Paris.
Iknayan, Herbert A., 1st Lt., 1414 7th St., Charleston.
Janson, Carl H., Capt., 17938 Homewood Ave., Homewood.
Jesser, Joseph H., Capt., 3358 N. Broadway, Chicago.
Johnson, Howard G., Capt., 210 S. 6th St., Casey.
Kaplan, Leo A., Capt., 808 Junior Terrace, Chicago.
Kaplan, Louis G., Capt., 3422 W. Monroe St., Chicago.
Karras, Samuel J., Capt., 105 N. 18th Ave., Melrose Park.
Kearns, Owen A., Capt., 321 W. State St., Rockford.
Koransky, Abraham R., Capt., 3816 Wrightwood Ave., Chicago.
Lattuada, Henry P., Major, 909 S. State St., Westville.
Lemberg, David A., Capt., 2900 W. Fitch Ave., Chicago.
Leventhal, Harry, Captain, 5425 Woodlawn, Chicago.
Lewin, Philip, Lt. Col., 1642 E. 59th St., Chicago.
Lustigman, Herman, Capt., 465 Grove St., Glencoe.
McBean, James B., Major, 7132 Ridgeland Ave., Chicago.
McCormick, Loyd J., Major, 2103 13th St., Moline.
McDougall, Francis J., Capt., 514 Williams St., River Forest.
McGinnis, William F., Capt., 318 W. Oak St., Carbondale.
McMorrow, Thomas R., Capt., 523 Prospect Rd., Peoria.
Maley, William F., Major, 44 N. Cherry St., Galesburg.
Magnelia, August L., Major, 1226 Garrison Ave., Rockford.
Maller, Adolph M., Capt., 4406 Clarendon Ave., Chicago.
Markiewicz, Simon V., Major, 4900 Pensacola Ave., Chicago.
Moore, Rollin S., Major, 306 Court St., Streator.
Nedoss, Hyman P., Capt., 1450 S. Kolin Ave., Chicago.
Olentine, Julie E., Capt., 3812 W. Monroe St., Chicago.
Pace, Anthony J., Capt., 734 N. Sawyer Ave., Chicago.
Parisi, Patrick, Capt., 1516 Edgewood Ave., Chicago Heights.
Pinne, George F., Capt., Billings Memorial Hosp., Chicago.
Plestina, Joseph M., Capt., 258 W. 33d St., Chicago.
Porte, David M., Capt., 3839 Ainslie St., Chicago.
Prunty, Francis C., Major, 1308 S. Race St., Urbana.
Puestow, Charles B., Col., 861 Lincoln, Highland Park.
Remich, Antone C., Capt., 12 W. 163d St., Calumet City.
Richter, John C. Jr., Capt., Presbyterian Hosp., Chicago.
Roe, Charles T., Capt., 544 S. Grove Ave., Oak Park.
Sandburg, Carl L., Capt., 405 Poplar St., Clinton.
Sargent, William F., Capt., 303 N. Broadway, Salem.
Saxon, Michael R., Capt., Oswego.
Shaw, Jesse W., Major, 107 Maple, Carbondale.
Steck, Irving E., Major, 5900 N. Christiana Ave., Chicago.
Sutton, Edmund B., 1st Lt., Halsted Rd., R. R. 6, Rockford.
Svetich, Edward M., Capt., 1128 N. Hickory St., Joliet.
Thompson, Harry G., Capt., 324 N. 12th, Mt. Vernon.
Turnbull, George C., Lt. Col., 636 Church St., Evanston.
Vinnecour, Max I., Major, 7227 Luella Ave., Chicago.
Wachtel, Hans, Capt., 5100 S. Cornell, Chicago.
Wallin, Bruce J., Capt., Rockford.
Walpole, Stewart H., Major, 305 East Ave., Park Ridge.
Wattleworth, Kent L., Capt., 324 W. Reynolds St., Newton.
Weinfeld, Gustave F., Capt., 351 Woodland Rd., Highland Park.
Wien, Norman A., Capt., 3833 Washington Blvd., Chicago.
Williamson, Holland, Col., 106 N. Vermillion St., Danville.
Wilson, Clyde S., Lt. Col., Commercial Bldg., Belleville.
Yarbrough, Charles L., Capt., 2613 Washington, Cairo.
Young, Richard H., Lt. Col., 2719 Broadway, Evanston.
Ziffren, Sidney E., Capt., 1323 4th Ave., Rock Island.
Zweig, Mitchell, Capt., 310 S. Hamlin Ave., Chicago.

Indiana

Anderson, Harry B., Capt., 2133 Bellefontaine St., Indianapolis.
Anderson, James W., Capt., 1165 N. Pershing Ave., Indianapolis.
Anderson, Walter C., Major, 380 S. 22d St., Terre Haute.
Applegate, Frederick M., Major, Corydon.
Ball, Joseph E., Capt., 1126 N. Tacoma Ave., Indianapolis.
Barnett, Ralph E., Major, 68½ E. 5th St., Peru.
Beaver, Howard W., Capt., 2203 S. Meridian, Indianapolis.
Berke, Robert D., Capt., Healthwin Hos., South Bend.
Booher, Norman R., Lt. Col., 104 E. Maple Rd., Indianapolis.
Brown, James C., Major, 458 Park Ave., Valparaiso.
Bruegge, Theodore J., Capt., 101 E. Markland, Kokomo.
Carney, John C., Capt., 613 W. Washington St., Monticello.
Chavinson, Benjamin, Capt., 222 S. 3d St., Decatur.
Clark, Cyrus J., Col., R. R. 14, Indianapolis.
Cogswell, Howard D., Major, 1620 Parkview, Whiting.
Comstock, Glenn E., Major, 504 Broadway, Gary.
Daggy, Benjamin T., Major, P. O. Box 255, Richmond.
Dingle, Paul E., Capt., 801 Peacock Rd., Richmond.
Donahue, George R., Major, 1619 S. 9th St., Lafayette.

PHYSICIANS SEPARATED FROM SERVICE

Indiana—Continued

Grosso, William G., Capt., 3502 Grand Blvd., East Chicago.
Hadley, David, Major, Indianapolis City Hosp., Indianapolis.
Hammond, Keith, Major, Paoli.
Hardesty, Kile C., Capt., 2536 Thompson Ave., Fort Wayne.
Harvey, Bennett B., Capt., Riley Hosp., Indianapolis.
Headley, Lloyd M., Capt., 205 S. East St., Lebanon.
Hemming, Philip C., Capt., 668 Addison St., Elgin.
Hurley, John R., Capt., Daleville.
Hynes, Roy T., Capt., 6176 N. Delaware, Indianapolis.
Jackman, Abraham I., Capt., 5618 Calumet Ave., Hammond.
Karpel, Bernard, Capt., % W. A. Kick, 1107 P St., Bedford.
Kendrick, Frank J., Major, 552 Johnson St., Garv.
Kidder, Orva T., Capt., Irene Byron Sanatorium, Fort Wayne.
Krieble, William W., Capt., 2320 Putnam Ave., Terre Haute.
Lett, E. Briscoe, Capt., Loogootee.
Love, George N., Capt., 819 Oak St., Connersville.
Lynch, Otis R., Capt., Marengo.
McDonald, Ralph M., Capt., R. R. 2, Mishawaka.
Michaelis, Stephen C., Capt., 4311 Marquette Dr., Fort Wayne.
Owen, Abraham M., Capt., 301 S. Brady St., Attica.
Palm, John M., Major, 27 E. Church St., Brazil.
Peacock, Norman F., Major, 7½ Mills Pl., Crawfordsville.
Picha, Valerian J., Capt., 89 9th St., Logansport.
Rabb, Harry S., Major, 1311 Union St., Indianapolis.
Reed, William C., Lt. Col., 1215 Atwater Ave., Bloomington.
Rommel, C. H., Major, 460 Northwestern Ave. W., Lafayette.
Sink, Frank G., Major, Remington.
Slick, Crystal R., Major, Lynn.
Smith, David L., Major, 723 Hume Mansur Bldg., Indianapolis.
Smith, Lawson F., Capt., 3802 N. Emerson Ave., Indianapolis.
Stone, David F., 1st Lt., Sunnyside San., Indianapolis.
Thompson, Charles F., Lt. Col., 6038 N. Olney, Indianapolis.
Vance, William C., Major, 200 S. 21st St., Richmond.
Walker, James S., Capt., 1402 N. Alabama, 215, Indianapolis.
Weldy, Bryce P., Capt., 121 E. Main St., Hartford City.
Wilson, Fred L., Capt., 1501 S. 3d St., Terre Haute.

Iowa

Andre, Gaylord R., Lt. Col., Lisbon.
Ashline, George H., Capt., 1010 N. 14th St., Keokuk.
Barnes, Charles A., Major, 206 E. Summit Ave., Shenandoah.
Barton, John C., Lt. Col., State Hosp., Independence.
Bastron, Harold C., Major, Red Oak.
Dushkin, M. A., Lt. Col., 602 Bankers Trust Bldg., Des Moines.
Flynn, Joseph E., Major, St. Univ. of Iowa Hosp., Iowa City.
Gantz, Albert J., Capt., Greenfield.
Geick, Raymond G., Capt., 709 Fowler St., Waterloo.
Howar, Bruce F., Major, Jewell.
Ihle, Charles W. Jr., Lt. Col., Cleghorn.
Jansonius, John W., Capt., Eldora.
King, Dean H., Capt., 915 E. 4th St., Spencer.
Longwell, Freeman H., Major, University Hosp., Iowa City.
Ludwick, Arthur L., Major, Waterloo.
Maire, Eugen J., Capt., Vail.
Milliard, Robert E., Lt. Col., 526½ Lake Ave., Storm Lake.
Marrs, Walford D., Capt., Box 146, Tabor.
Morrison, Roland B., Capt., 117 E. 6th St., Carroll.
Mosher, Martin L. Jr., Capt., West Branch.
Needles, Roscoe M., Capt., Atlantic.
Newton, Isaac J., Capt., University Hosp., Iowa City.
Nicoll, Charles A., Major, Panora.
Pascoe, Paul L., Capt., 1127 N. Adams, Carroll.
Paulus, Edward W., Lt. Col., 1039 E. College St., Iowa City.
Quinn, Francis P., Major, 887 Mt. Carmel Rd., Dubuque.
Shapiro, Seymour I., Capt., Miss. Hotel, Davenport.
Steffey, Frederick L., Major, 703 Blontleau St., Keokuk.
Willett, Wilton J., Capt., Manclester.
Wilson, Frederick D., Capt., Morning Sun.

Kansas

Aldis, John, 1st Lt., 1014 Merchant St., Emporia.
Bartlett, Wayne C., Major, 106 N. Main St., Wichita.
Filkin, Lawrence E., Lt. Col., 721 W. 6th St., Junction City.
Gonser, Karl B., Capt., 1300 S. 4th St., Leavenworth.
Lewis, George K., Lt. Col., 801 N. 6th St., Garden City.
Mandeville, George, Major, Crown Bldg., Dodge City.
Overholser, Norman H., Capt., 115½ S. Main St., El Dorado.
Rabe, Melvin A., 1st Lt., Kanas Univ. Hospital., Kansas City.
Schwartz, Willard C., Major, 1919 Poyhtz, Manhattan.
Sutherland, Earl W. Jr., Capt., Burlingame.
Voorhees, Gordon S., Capt., Univ. of Kansas, Kansas City.

Kentucky

Amos, B. K., Capt., Princeton.
Barnard, William H., Lt. Col., Elizabethtown.
Barr, Robert H., Col., 405 St. Ann St., Owensboro.
Bate, Richard A. Jr., Capt., 2202 Sheffield Blvd., Louisville.
Bell, Joseph C., Lt. Col., Glenview, Louisville.
Bingham, Roy E., Capt., Box 322, Route 6, Louisville.
Booth, Thomas E., Major, Louisville Citv Hosp., Louisville.
Brown, Leonidas X., 1st Lt., Williamsburg.
Chumley, Jack L., Major, Louisville City Hosp., Louisville.
Clifton, Wilburn P., Capt., 234 Sycamore St., Barbourville.
Coons, Albert H., Major, 132 1st Ave., Gloversville.
Cowan, Albert W., Capt., N. 25th St., Middlesboro.
Eckenhoff, James E., Capt., Good Samaritan Hosp., Lexington.
Gilbert, James T. Jr., Major, 953 Magnolia, Bowling Green.
Gray, Laman A., Major, 610 Upland Rd., Louisville.
Hall, Charles N., 1st Lt., 301 W. Mount Vernon St., Somerset.
Hancock, James D., Lt. Col., 80 Valley Rd., Louisville.
Heitz, Raymond, Major, 1732 Deer Lane, Louisville.
Holt, Howard P., Capt., Twila.
Jakeman, Harry A., Capt., W. Main St., Stanford.
Johnson, James E., Lt. Col., Jenkins.
McIlvoy, Daniel B. Jr., Major, Grundy Ave., Springfield.
Murray, Hershell B., Capt., West Liberty.
Nichols, William E., Capt., Manchester.
Noble, Vernon A., Capt., 3614 Del Park Terrace, Louisville.
Pennington, Clarke P., Capt., 2211 Glenmary Ave., Louisville.
Rompf, John H., Lt. Col., % Eastern State Hosp., Lexington.
Stevenson, Richard V., 1st Lt., 120 Iroquois Ct., Lexington.
Weddle, Richard H., Major, 133 N. Maple St., Somerset.
Work, C. E., Lt. Col., 122 N. Ft. Thomas Ave., Fort Thomas.

Louisiana

Abramson, Paul D., Major, 808 Jordan St., Shreveport.
Atkinson, Robert H., Capt., 3750 Youree Dr., Shreveport.
Bishop, Clarence A., Lt. Col., 986 Harding Dr., New Orleans.
Browning, William H., Capt., 1717 Marshall St., Shreveport.
Broyles, Joe E., Major, 3936 Baltimore St., Shreveport.
Cabiran, Louis R., Capt., 5902 Dauphine St., New Orleans.
Campbell, Nicholas M., Capt., Mercy Hosp., New Orleans.
Chalstrom, Harry E. Jr., 1st Lt., 2202 Gen. Pershing St., N. O.
Chavez, Stephen A., Capt., 2317 Broadway Ave., New Orleans.
Davidge, Lucious L., Major, 940 Margaret Place, Shreveport.
Deddens, Lloyd E., Capt., Apt. 5B Groves St., DeRidden.
Edwards, Arthur M., Major, Bernice.
Ellington, Joseph C., Major, Alto.
Everett, Peter 3d, Capt., 900 Independence St., New Orleans.
Gahagan, Harry Q., 1st Lt., Coushatta.
Guidry, Edwin R., Major, 720 Broadway, New Orleans.
Gwin, John V., Major, 7031 Freret St., New Orleans.
Hardy, W. R., Lt. Col., 626 Maison Blanche Bldg., New Orleans.
Ilgenfritz, Hugh C., Major, 2636 Napoleon Ave., New Orleans.
LaNasa, James J., Major, Charity Hosp., New Orleans.
Landry, Joseph D., Capt., 1112 Union Bldg., New Orleans.
Lorenzen, Lee H., Capt., 1636 Broadway, New Orleans.
McCook, Walter W. Jr., Major, 911 Wilkinson St., Shreveport.
Meyer, Harry, Major, 2231 Calhoun St., New Orleans.
Nelken, Bernard E., Major, 2027 Kleinert Ave., Baton Rouge.
Nix, James T., Capt., 2140 Carrollton Ave., New Orleans.
Pardue, Cleveland H., Capt., Vivian.
Pasternack, Morris, Major, Charity Hosp., New Orleans.
Rand, Paul K., Major, Vance Ave., Alexandria.
Rizzo, Frank P., Lt. Col., 320 Grammont St., Monroe.
Rosen, Warren L., Major, 7030 Birch St., New Orleans.
Rougelot, Robert E., Major, 2737 Ursuline Ave., New Orleans.
Rozier, John S. III, Capt., Leesville.
Sabatier, Joseph A. Jr., Capt., Eunice.
Shushan, Morris, Major, 8232 Pritchard Pl., New Orleans.
Storck, A. H., Lt. Col., 1458 Nashville Ave., New Orleans.
Treadway, James L. Jr., Capt., 38 Lousiana Ave., Alexandria.
Troxler, Ferdinand A., Major, Reserve.
Vincent, Francis J., Capt., Marksville.
Weilbaecher, J. O. Jr., Lt. Col., 5826 Clara St., New Orleans.
Wexberg, Leopold E., Major, 1233 Nashville, New Orleans.
Winder, Paul W., Major, Highland Clinic, Shreveport.
Vasquez, Guillermo, 1st Lt., Franklin.
Woodhouse, Keith W., Col., Vinton.

Maine

Greene, Merrill S. F., Major, 466 Main St., Lewiston.
Howard, Henry M., Major, 105 Franklin St., Rumford.
Johnson, Albert C., Capt., 90 Prospect St., Portland.
Proctor, Thomas E., Capt., 8 McKnown St., Boothby Harbor.
Sumner, Charles M., Capt., West Sullivan.

PHYSICIANS SEPARATED FROM SERVICE

Maryland

Bordley John E., Lt. Col., 319 Tunbridge Rd., Baltimore.
Brack, Charles B., Major, 302 Underwood Ct., Baltimore.
Callahan, Timothy A., Capt., Bel Air.
Cohen, Lawrence J., Major, 2510 Keyworth Ave., Baltimore.
Cutts, Morgan, Major, 211 Wendover Rd., Baltimore.
Dickey, Francis G., Lt. Col., 600 Winans Way, Baltimore.
Fielding-Reid, F., Capt., Maryland Club, 1 E. Eager, Baltimore.
George, Joseph M., Capt., Sudersville.
Goodman, Howard, Lt. Col., 511 Wilton Rd., Towson.
Harvey, Abner M., Major, Johns Hopkins Hosp., Baltimore.
Henning, Emil H. Jr., Capt., 601 Winans Way, Baltimore.
King, John T., Col., 219 W. Lanvale St., Baltimore.
Lambert, Richard H., Lt. Col., Johns Hopkins Hosp., Baltimore.
Mays, Howard B., Major, 4313 Marble Hall Rd., Baltimore.
Quinn, William C., Capt., Main St. Ext., Crisfield.
Revell, Samuel T. R. Jr., Capt., 3814 Fernhill Ave, Baltimore.
Shapiro, Abraham A., Major, 6711 Park Hgts. Ave., Baltimore.
Stafford, E. S., Lt. Col., 3722 Tudor Arms Ave., Baltimore.
Strong, Paul S., Major, Sydenham Hosp., Baltimore.
Vollmer, Donald H., Major, Wash. Sanitarium, Takoma Park.

Massachusetts

Adams, Ronald W., Capt., 132 Homer, Newton.
Ainsworth, Leonard B., Capt., 351 Essex St., Lawrence.
Andren, Henry E., Major, 45 W. Newton St., Boston.
Aufrance, Otto E., Major, 374 Commonwealth Ave., Boston.
Axelrod, George, Major, 302 Chestnut St., Clinton.
Bartlett, Marshall K., 1st Lt., 43 Chestnut St., Dedham.
Betts, Reeve H., Major, 51 Devoy Rd., Newton Center.
Bland, Edward F., Lt. Col., Mass. Gen. Hosp., Boston.
Bloomenthal, Abraham P., Capt., 7 Banks St., Waltham.
Budington, Harold F., Lt. Col., 958 State St., Springfield.
Churchill, Edward D., Col., 269 Prospect St., Belmont.
Clifford, Milton H., Lt. Col., 17 Lowell St., Camridge.
Curtis, Sprague, Capt., Westfield State Hosp., Westfield.
Dibbins, Samuel A., Major, 528 Andover St., Lowell.
Dickson, William A., 1st Lt., Watson.
Dowd, George C., Capt., 87 Norfolk St., Worcester.
Ellis, Daniel S., Capt., Mass. GH Fruit St., Boston.
Farren, Edward B., Capt., 108 Moraine St., Brockton.
Fish, James E., Col., Melrose.
Flake, Carlyle G., Lt. Col., 9 Bryon St., Boston.
Ford, Richard, Major, 9 Riedesel Ave., Cambridge.
Galuszka, Bronislaus A., Capt., 895 Front St., Chicopee Falls.
Goldberg, George, Capt., 138 Tappan St., Brookline.
Greenfield, Irving, Capt., 41 Burbank St., Boston.
Gregg, Ward I., Major, Gerry's Landing, Cambridge.
Gregory Harold E., Capt., 34 Washington St., Beverley.
Halsted, James A., Major, 759 High St., Dedham.
Harris, Casimir P., Capt., 21 Duffield Rd., Auburndale.
Hart, Leon M., Capt., 94 Washington St., Weymouth.
Hovenanian, Michael S., Capt., 97 Jackson St., Cambridge.
Kabler, Paul W., Major, 127 Washington St., Brighton.
Karp, Isadore A., 1st Lt., 153 Shurtleff St., Chelsea.
Kasparian, Karl D., Lt. Col., 310 Talbot Ave., Dorchester.
Kelley, Sylvester B., Major, 34 Exeter St. W., Newton.
King, Donald S., Col., 111 Holland Rd., Brookline.
Kranes, Alfred, Major, 38 Carleton Circle, Belmont.
Lasalle, Arthur, Capt., 38 Rock St., Fall River.
Lawrence, Knowles B., Capt., 171 Bay State Rd., Boston.
LeBeaux, Lincoln, Capt., Shrewsbury.
Leighton, L. H., Major, 1802 Massachusetts Ave., Cambridge.
Levine, Julius, Capt., 253 Norfolk St., Dorchester.
Mahoney, Frank A. Jr., Major, 16 Harriet St., Springfield.
Maietta, Angelo L., Capt., 408 Main St., Winchester.
Myers, Jack D., Lt. Col., 721 Huntington Ave., Boston.
Meltzer, Adolph, Capt., 233 May St., Worcester.
O'Hara, John L., Capt., 136 Hunnewell Ave., Newton.
Parsons, Langdon, Major, 57 Dudley Rd., Newton Center.
Press, Harry A., Capt., 598 Tyler St., Pittsfield.
Rutledge, David I., Major, 605 Commonwealth Ave., Boston.
Sahler, Otto D., Major, Mass. Gen. Hosp., Boston.
Sarris, Spiros P., Major, 152 School St., Lowell.
Schwartz, Harry H., Major, 562 Broadway, Everett.
Settlage, Arnold F. E., Capt., 244 High St., Newburyport.
Shaughnessy, John R., Major, 24½ Winter St., Salem.
Sisson, Milton M., Capt., 750 Barnardston Rd., Greenfield.
Sneiderman, Robert, Capt., 1 Atlantic St., Lynn.
Staples, Oscar S., Major, 136 Milton Ave., Hyde Park.
Suby, Howard I., Major, 137 Englewood Ave., Brookline.
Thomas, Edwin B., Capt., Fitchburg.
Thompson, Leonard B., Capt., 346 Elm St., Gardner.
Ulfelder, Howard, Capt., 27 Shakan Rd., Belmont.

Massachusetts—Continued

Verstandig, Charles C., Major, 220 Chelsea St. E., Boston.
Wallace, W. M., Capt., Children's Hosp., 300 Longwood, Boston.
Weitz, Mark, Capt., 1307 Commonwealth Ave., Allston.
Welch, William J., Capt., 119 Winthrop St., Taunton.
Whitcomb, Austin E., Capt., 21 Bardwell St., Hadley Falls.
Yens, Otto C., Lt. Col., 22 Forest St., Cambridge.
Zoll, Paul M., Major, 25 Peterborough St., Boston.

Michigan

Andrews, Nelson A. C., Capt., 310 E. Main St., Flushing.
Bader, Benjamin H., Lt. Col., 3281 Pasadena, Detroit.
Belanger, William G., Capt., 92 Orchestra Pl., Detroit.
Boccaccio, John L., Capt., 5054 Van Dyke, Detroit.
Boucher, Roman E., Capt., 2830 Glenwood Ave., Royal Oak.
Brown, Robert W., Capt., Detroit.
Buechner, Frederick W., Major, 100 Guest St., Battle Creek.
Carstensen, Vincent H., Capt., U. S. Marine Hosp., Detroit.
Chesluk, Herman M., Capt., 1983 Taylor, Detroit.
Chipman, Elwood M., Capt., 47 E. Chicago St., Quincy.
Claxton, Wilbert T., Capt., Britton.
Conger, Kyril B., Major, Univ. Hosp., Ann Arbor.
Corgill, Donald A., Major, 3079 W. Grand Blvd., Detroit.
Delbert, Stewart G., Capt., Wyandotte Gen. Hosp., Wyandotte.
De Vel, Leon, Lt. Col., 739 Plymouth Blvd. S. C., Grand Rapids.
Dorland, John A. C., Capt., 17 Court St., Lapeer.
Feldkamp, Lee E., Major, 508 Brower St., Ypsilanti.
Feldman, Paul H., Capt., 15036 Muirland St., Detroit.
Fellows, Kenneth E., Capt., 1238 Alexander S.E., Grand Rapids.
Flint, Charles H., Major, Hart.
Florentz, Theodore R., Capt., 8848 Buhl Ave., Detroit.
Free, Harry W., Col., Grace Hosp., Detroit.
Frohlich, Moses M., Lt. Col., Univ. Hosp., Ann Arbor.
Gillespie, S. M., Capt., 25313 Ingleside, RFD 5, Birmingham.
Gowen, Guy H., Lt. Col., 1101 Baldwin Ave., Ann Arbor.
Gutman, Emil, Capt., 2078 Mill St., Lincoln Park.
Hansen, Carl M., Capt., 205 Bellvue, Stanton.
Harley, Garth H., Capt., 7620 Kipling, Detroit.
Harryman, James E., Capt., 118 S. Washington Ave., Lansing.
Horowitz, Samuel F., Major, 620 N. Madison St., Bay City.
Huff, Ralph H., Capt., Receiving Hosp., Detroit.
Husted, Franklin P., Major, 803 N. McLellan St., Bay City.
Jones, Tyre K., Capt., 118 W. Green, Marshall.
Kavanaugh, William R., Capt., 442 Stuart Ave., Kalamazoo.
Kitzmiller, John L., Capt., 68 E. Willis, Detroit.
Knaggs, Earl J., Capt., 13179 Phelps Blvd., Wyandotte.
Konopa, John F., Major, Manistee.
Kuhs, Milton L., Capt., 2755 Bronson Blvd., Kalamazoo.
Lauppe, F. A., Major, 155 Elmhurst Ave., Highland Park.
Lemen, Charles E., Capt., 521 Washington St., Traverse City.
Lindenfeld, Frederick H., Capt., 611 State St., St. Joseph.
Lipschutz, Louis S., Major, Eloise Hosp., Eloise.
Lowell, Vivion F., Major, Ypsilanti State Hosp., Ypsilanti.
McColl, Charles W., Capt., Wyandotte.
McNickle, Jerry H., 1st Lt., 1303 Delaware Ave., Detroit.
MacIntyre, D. S., Lt. Col., 714 Linda Vista Ave., Ann Arbor.
Meier, Harold J., Major, 70 N. Hudson St., Coldwater.
Merritt, Harry E., Capt., 6533 16th St., Detroit.
Miller, Harry, Major, 1130 Parker Ave., Detroit.
Miller, K. T., Lt. Col., 746 E. Division St., Cadillac, Wexford.
Mosier, Dwight J., Capt., 307 W. Midland St., Bay City.
Muehlig, George K., Capt., 1604 Grauger Ave., Ann Arbor.
Ogilive, Gordon D., Capt., 255 1st St., Manistee.
Ott, Harold A., Major, 19511 Shresbury Rd., Detroit.
Pauli, Theodore H., Lt. Col., 161 Illinois, Pontiac.
Pearson, Stanley M., Capt., 501 W. Jenny, Bay City.
Phillips, Francis J., Capt., Herman Kietev Hosp., Detroit.
Prentice, Edwin W., Capt., 22213 Olmstead Ave., W. Dearborn.
Purves, William L., Major, 222 E. 1st St., Flint.
Quarton, Albert E. Jr., Capt., University Hosp., Ann Arbor.
Ross, Hyman, Capt., 2676 Webb, Detroit.
Richards, Frank D., Capt., DeWitt.
Rogers, John D., Lt. Col., 1232 W. Maple St., Adrian.
Rowell, Wilfred J., Capt., 18633 Puritan Ave., Detroit.
Schlecte, Irwin C., Major, 326 Walnut, Rochester.
Shepherd, Walter F., Capt., Ross Apt. 5, Owosso.
Smith, Fred R., Capt., 17501 Woodinham Dr., Detroit.
Tofteland, Elmer H., Capt., 902 Woodside Dr., Flint.
Vail, Harry F., Capt., 408 W. Jenny St., Bay City.
Watson, Bernard A., Lt. Col., 114 E. Locust St., Three Oaks.
Wheeler, Stewart C., Capt., 17554 Ohio, Detroit.
Wright, Leonard D., Capt., Rt. 2, Box 50, Jackson.
Zbudowski, Alexander S., Capt., 3400 Evaline Ave., Hamtramck.
Zinn, August K., Capt., 55 Post Rv., Battle Creek.

PHYSICIANS SEPARATED FROM SERVICE

Minnesota

Aling, Charles A., Major, 2920 Victory Mem. Dr., Minneapolis.
Barker, John D., Major, 37 Wetzel St., Warren.
Beek, Harvey O., Lt. Col., St. Paul.
Belzer, Meyer S., Lt. Col., 2915 40th Ave., South Minneapolis.
Benjamin, Edwin G., Lt. Col., 2202 Pleasant Ave., Minneapolis.
Black, Earl J., Capt., Commodore Hotel St., St. Paul.
Bloom, Norman B., Major, Minneapolis.
Butter, John R., Capt., 6630 Normandale Rd., Minneapolis.
Caldwell, Hayes W., Capt., 225 4th Ave., Rochester.
Clapp, Stewart, 1st Lt., 5319 Oakley St., Duluth.
Chalmers, James H. Jr., Major, Madison.
Clarke, Edward T., Capt., Mayo Clinic, Rochester.
Clarke, William O., Major, 1929 7th Ave., Hibbing.
Emond, Joseph S., Major, Farmington.
Geissler, G. A., Major, V. C. C. Co., 2713 Cp. NP-1 Hinckley.
Goehrs, Gilman H., Lt. Col., 1008 8th Ave. S., St. Cloud.
Hankerson, Robert G., Capt., Minnesota Lake.
Hanley, James L. Jr., Major, Mayo Clinic, Rochester.
Hartmann, Clarence M., Major, Fairfax.
Hartman, Jack, Capt., 704 15th Ave. S.E., Minneapolis.
Hertz, Myron J., Capt., 1153 Summit Ave., St. Paul.
Holland, Cleon R., Major, Rochester.
Howe, Rulon F., Lt. Col., 210 6th St. S.W., Rochester.
Hultkrans, Rudolph E., Capt., 4823 Dupont Ave. S., Minneapolis.
Hunt, Robert S., Major, Fairmont.
Johnson, John W., Major, Kerkhoven.
Kaasa, Laurin J., Capt., Albert Lea.
Kapsner, Alfred T., Capt., Pierz.
Katzovitz, Hyman, Capt., 158 Eaton Ave., St. Paul.
Kinports, Edward B., Capt., 928 3d St., International Falls.
Knutson, Lewis A., Capt., Belgrade.
Lind, Carl J. Jr., Lt. Col., 3104 45th Ave. S., Minneapolis.
McGroarty, Brian J., Major, Easton.
Meyer, Wallace M., Capt., Rollingstone.
Mitchell, Mancel T., Major, Minneapolis.
Moir, William W. Jr., Major, 316 W. 50th St., Minneapolis.
Murray, Robert A., Lt. Col., Aitkin.
O'Brien, John P., Lt. Col., The Mayo Foundation, Rochester.
O'Leary, John H., Capt., 509 7th St. N., Staples.
Plotke, Harry L., Capt., 1st Natl. Bank Bldg., Little Falls.
Polski, Paul G., Capt., 914 Wakefield Ave., St. Paul.
Selin, Goldon, Capt., Oak Terrace.
Shima, George J., Capt., Montgomery.
Strickler, Jacob H., Capt., Brainerd.
Teisberg, John E., Capt., Middle River.
Wasson, Loren F., Capt., Alexandria.

Mississippi

Armstrong, James H., Lt. Col., Oxford.
Brackstone, LeRoy B., Capt., Iuka.
Burt, Lawrence W., Major, 826 5th Ave., Laurel.
Davis, Jesse T., Lt. Col., Speedway, Corinth.
Feemster, Lucien C., Major, 321 N. Green St., Tupelo.
Fogel, Morris, Lt. Col., Vet. Administration Facility, Biloxi.
Gilding, Joseph P., Capt., 145 N. Main St., Vicksburg.
Hall, William M., Major, % Mrs. L. B. Robinson, Centreville.
Henry, Eugene M., Capt., Charleston.
Jackson, Claude A., Capt., 111 N. Wells St., Kosciusko.
Jackson, Paul, Major, Liberty.
Laird, Earl L., Major, Union.
McAmis, Elton L., Capt., Port Gibson.
Massengill, Frank C., Capt., 321 W. Chippewe St., Brookhaven.
Moore, Daniel H. Sr., Capt., 2506 29th Ave., Meridian.
Murfee, John A., Major, Box 338, Amory.
Pearce, Leroy S., 1st Lt., Falkner.
Pritchard, Clarence D., Major, Marks.
Purser, Thomas Jr., Major, McComb.
Snelling, Murdock M., Major, 712 E. Beach, Gulfport.
Ward, Charles E., Major, Jackson.
Watkins, Charles, Capt., Shaw.
Williams, Howard S., Major, Hattiesburg.
Williams, Thomas R., Capt., Batesville.
Witte, Wallie S., Capt., 201 Washington St., Leland.

Missouri

Aaron, George, Capt., 5831 Rockhill Rd., Kansas City.
Alvis, Edmund B., Major, 308 N. 6th St., St. Louis.
Arrowsmith, William R., Capt., 4539 Parkview Pl., St. Louis.
Backlar, Joseph, Major, 1025 McKnight Rd., Richmond Hgts.
Barnes, Seth S., Major, Owensville.
Bartlett, Robert W., Lt. Col., 607 N. Grand Ave., St. Louis.
Burford, T. H., Major, 600 S. Kingshighway St., St. Louis.
Carey, William A. Jr., Capt., St. Louis City Hosp., St. Louis.

Missouri—Continued

Chastain, Maurice W., Capt., Kansas City.
Cox, Kenneth E., Capt., 45 E. 55th Terr., Kansas City.
Curran, Desmond, Capt., 925 Grand Ave., Kansas City.
Donley, Robert R., Capt., 633 Atlanta, Webster Groves.
Darnell, Thomas F. B., Major, Macon.
Dowd, James F., Capt., 7220 W. Park Ave., Richmond Heights.
Eberhard, Theodore P., Col., 801 W. Ash St., Columbia.
Ellis, Coburn H., Capt., Sweet Springs.
Elson, Julius, Capt., 7330 Pershing St., St. Louis.
Evans, Ezra L. Jr., Lt. Col., 1103 Cherry St., Springfield.
Freedman, Harold, Major, 7246 Pershing Ave., University City.
Gamet, Joseph H., Lt. Col., 508 W. 8th St., Rolla.
Hogue, Frank S., Major, 103 Ward Parkway, Kansas City.
Hollo, Vencel W., Capt., Firmin Desloge Hosp., St. Louis.
Hopkins, Florian G., Major, Box 506, Gideon.
Kelley, Gilbert B., Major, 1010 Wakefield Ave., Savannah.
Kitchen, William M., Major, Woodland Hosp., Moberly.
Kimes, Ira D., Capt., 403 S. Cherry, Cameron.
Klein, Adelbert H., Major, 2632 S. Kingshighway, St. Louis.
Kleykamp, Elmer A. Jr., Capt., 3628 Shaw Ave., St. Louis.
Kotner, Lawrence M., Capt., 6610 Enright Ave., Univ. City.
Leech, Charles A. Jr., Major, 718 Maryland, Columbia.
Lo Piccolo, V. J., 1st Lt., 73 Lake Forest, Richmond Heights.
McKinstry, Karl V., Capt., 105 S. 4th St., DeSoto.
Mulligan, Leo V., Major, City Hosp. No. 1, St. Louis.
Myers, Daniel W., Lt. Col., 5653 Waterman St., St. Louis.
Myers, Robert M., Capt., 222 W. 67th St., Kansas City.
Pareira, M. D., Major, Ellis Fischel State Cancer H., Columbia.
Parker, Joe M., Major, Ellis Fischel Hosp., Columbia.
Patton, John F., Lt. Col., 723 University Club Bldg., St. Louis.
Robinson, George G., Capt., Humansville.
Schwartz, Eugene J., Capt., 833 S. Weller, Springfield.
Stockwell, A. L., Major, 625 Professional Bldg., Kansas City.
Vandover, John T., Major, 3728 Arsenal St., St. Louis.
Wedig, John H., Major, Barnes Hosp., St. Louis.
Werner, William A., 1st Lt., 5573 Cates St., St. Louis.
Wyatt, Lisle M., Capt., 4905 Holly St., Kansas City.

Montana

Bogardus, George M., Capt., 31 5th Ave., E. Kalispell.
Donich, George M., Major, St. James Hosp., Butte.
Eck, Raymond L., Capt., 612 Montana Bldg., Lewiston.
Malee, John J., Capt., 215 Walnut St., Anaconda.
Pearson, John A., Capt., Park Hotel Bldg., Livingston.

Nebraska

Campbell, Louis S., Major, 1602 Broadway, Scottsbluff.
Civin, W. Harold, 1st Lt., 1412½ 24th St., Omaha.
Crynes, Sylvester F., Major, 2010 N. 49th St., Omaha.
Foote, Donovan B., Capt., Foote Bldg., Hastings.
Grabow, Henry C., Major, 120 S. 44th St., Omaha.
Hankins, Charles R., Capt., 5121 Erskine St., Omaha.
Hervert, James W., Capt., Valparaiso.
Kelley, William E., Capt., 4303 Woolworth Ave., Omaha.
Lipp, Frank E., Major, 119 Chestnut St., Kimball.
MacKenbrock, Frederick C., Lt. Col., Hotel Rome, Omaha.
O'Donnell, Reynolds J., Capt., 2208 16th St., Columbus.
Rich, Edward L., Major, Grand Island.
Shamberg, Alfred H., Major, University Hosp., Omaha.
Teply, Gene L., Capt., Howells.
Young, George A. Jr., Lt. Col., 5228 Jones St., Omaha.

New Hampshire

Bennert, Harry W., Capt., 1485 Belmont St., Manchester.
Hagerty, Edward D., Major, 4 Broad St., Nashua.
Hubbard, Edward O. Jr., Capt., R. F. D. 1, Peterborough.
McLeod, John K., Capt., 23 Elm St., Penacook.
March, Luther A., Capt., 31 Berkeley St., Nashua.
Miller, John H., Capt., 1388 N. Main St., Laconia.
Milliken, Nathan T., Capt., Hitchcock Clinic, Hanover.
Willson, Allan T., Major, Farmington.
York, Charles L. Jr., Capt., 18 Winter St., Plymouth.
Yudickey, Stanley W., Capt., 573 Spruce St., Manchester.

New Jersey

Amato, Romelo J., Capt., 370 LaFayette St., Newark.
Baker, Banks S., Capt., 618 Benson St., Camden.
Bar, Samuel, Major, 54 Main St., Englistown.
Barbour, George E., Capt., 118 W. High St., Somerville.
Barlow, George B., Capt., 157 Engle St., Englewood.
Bayer, Eric C., Capt., 237 Franklin Ave., Cliffside Park.
Berenson, Samuel J., Major, 1012 E. Jersey St., Elizabeth.
Bettle, Ronald A., Major, Hackensack Hosp., Hackensack.

PHYSICIANS SEPARATED FROM SERVICE

New Jersey—Continued

Beyer, William, Lt. Col., 612 Undercliff Ave., Edgewater.
Block, Milton, Capt., 342 Union Ave., Irvington.
Board, Thomas P., 1st Lt., 294 Easton Ave., New Brunswick.
Bonanno, Peter J., 1st Lt., 500 79th St., North Bergen.
Bookrajian, Edward N., Capt., 8027 Hudson Blvd., N. Bergen.
Booth, William K., Major, 304 William St., Boonton.
Boquist, Walter A., Major, 380 Hudson St., Phillipsburg.
Boylan, Mathew E., Capt., 2690 Blvd., Jersey City.
Brauer, Selig L., Capt., 2012 Blvd., Jersey City.
Burnett, Hayes J., Capt., 1 Hawthorne Pl., Montclair.
Cassio, William L., 1st Lt., 125 Elm St., Montclair.
Clark, Morris B. Jr., 1st Lt., 124 Roberts Ave., Haddonfield.
Cooney, Raynald T., Capt., St. Francis Hosp., Trenton.
Copleman, H. B., Capt., 111 Livingston Ave., New Brunswick.
Cox, William T. R., Major, 345 S. Broad St., Elizabeth.
Degenhardt, Ira H., Major, 114 S. 1st Ave., Highland Park.
Dein, Harry L., Capt., 4 S. Plaza Pl., Atlantic City.
DeSantis, Orazio J., Capt., 100 N. 2d St., Millville.
Dimun, John T. Jr., Major, 960 S. Broad St., Trenton.
English, Harrison F. III, Major, 10 Morningside Dr., Trenton.
Enion, George A., Col., Sea Girt.
Franklin, H. J., Capt., Staff House, Med. Center, Jersey City.
Freeman, Ray M., Lt. Col., 922 Orchard Terrace, Linden.
Freyberger, George A., Capt., 36 Bonn Pl., Weehawken.
Gilligan, Walter W., Major, Nutley.
Glass, Harry L., Capt., 1009 Park Ave., Plainfield.
Gordon, Norman W., Capt., 71 Cleveland Ave., Elizabeth.
Grandin, Dean J., 1st Lt., 1073 Anderson Ave., Palisade.
Greenberg, Solomon, Capt., 119 W. 28th St., Bayonne.
Hagen, Walter H., Major, 671 Lincoln Ave., Orange.
Harbeson, James P. III, Capt., 1213 Eldridge W., Collingswood.
Hardy, John W., Capt., 53 Main St., Farmingdale.
Hayman, Irving R., Capt., 681 Broadway, Paterson.
Heyman, Arthur, Major, 79 Baldwin Ave., Newark.
Hillsman, Robert B., 1st Lt., 681 Queen Anne Rd., Teaneck.
Hollins, George G., Capt., 71 Cates Ave., Montclair.
Hughes, Joseph F., Capt., 16 N. Broad St., Woodbury.
Jones, William F., Capt., 1318 Broad St., Pleasantville.
Joyner, Austin, Lt. Col., 27 Vincent St., Hillsdale.
Kaycoff, Aaron J., Capt., 1063 North Ave., Elizabeth.
Kollar, Charles S., Capt., 131 Lincoln Ave., Ridgewood.
Kuder, Joseph M., Lt. Col., 104 Garden St., Mt. Holly.
Kunderman, Philip J., Major, 215 Harper St., Highland Park.
Laudig, Guy H., 1st Lt. 361 Speedwell Ave., Morris Plains.
Lavine, Samuel C., Capt., 88 Livingston Ave., New Brunswick.
Leach, John E., Lt. Col., 600 14th Ave., Paterson.
Legato, Samuel F., Capt., 417 Palisade Ave., Cliffside Park.
Levine, Harold N., Capt., Hosp. for Mental Diseases, Secaucus.
Long, John F., Capt., 205 N. 4th St., Harrison.
Longo, James J., Capt., 74 Ridge Rd., North Arlington.
Lippman, Harold E., Capt., 108 Elizabeth Ave., Newark.
Luce, Henry A., Capt., 158 Hickory St., Orange.
McDonnell, George J., Capt., 80 W. Main St., Freehold.
Magnes, Max, Major, 271 Park Ave., Paterson.
Matthews, Edward J., Capt., 224 12th Ave., Newark.
Mazur, Edward F., Capt., 1255 Liberty St., Camden.
Merlo, Frank V., Capt., 33 Prince St., Elizabeth.
Miller, Clark F., Capt., St. Peter's Hosp., New Brunswick.
Monaco, Saverio A., Capt., 293 Camden St., Newark.
Monaloy, Morris A., Capt., 24 Day St., Clifton.
Moriconi, Albert F., Major, 438 Hamilton Ave., Trenton.
Naidorff, Saul A., Capt., 421 W. 7th St., Plainfield.
Netz, Lester W., Capt., 365 W. Anderson St., Hackensack.
Ney, Julian M., Capt., 443 Baldwin Rd., Maplewood.
Nussbaum, Harvey E., Major, 13 Lewis Dr., Maplewood.
Oatman, Jack G., Major, Greystone Park.
Pauly, Arthur N., Capt., 105 Willowdale Ave., Montclair.
Perrone, Arthur F., 1st Lt., 522 52d St., West New York.
Pierce, Henry A., Major, 465 Broad Ave., Leonia.
Pino, Anthony, Major, 196 Irving Ave., Bridgeton.
Pomeranz, Raphael, Major, 31 Lincoln Park, Newark.
Price, Kasper J., Major, 111 6th St., Elizabeth.
Rachlin, Harry T., Capt., 82 Schuyler Ave., Newark.
Reinhardt, Warren I., Capt., 276 Springdale Ave., East Orange.
Restaino, Charles F., Major, 465 Parker St., Newark.
Richter, D. A., Capt., % C. A. Richter, 184 Demarest, Englewood.
Roberts, Montague A., Capt., 124 Linden Ave., Englewood.
Rosenthal, Sydney, Capt., 88 Pomona Ave., Newark.
Sackin, Stanley, Capt., 1009 Hamilton Ave., Trenton.
Shavelson, Irving C., Capt., 3822 Ventnor Ave., Atlantic City.
Stankiewicz, Florian S., 1st Lt., 240 Union St., Jersey City.
Sussman, Harold, 1st Lt., 541 44th St., Union City.
Tatem, Henry R. Jr., Major, Pine & Atlantic Ave., Audubon.
Thompson, James H., Capt., Upper Montclair.
Virgilio, Anthony A., Capt., 87 S. Centre St., Orange.
Wangner, William F., Major, 20 Warren St., Bloomfield.
Waterman, Samuel M., Major, 364 Clinton Ave., Newark.
Wechtel, Karl N., Capt., 726 Central Ave., Woodmer.
Weinstock, Michael B., Major, 13 Hillside Ave., Newark.

New Mexico

Lewis, Romeo H., Capt., 412 E. Lead Ave., Albuquerque.
Owens, Ross, Capt., Box 355, Hobbs.
Pike, William W., Major, P. O. Box 97, Expanola.
Rice, Lucien G. Jr., Capt., 100 S. Arno St., Albuquerque.

New York

Abramson, Harold A., Major, 315 E. 68th St., New York.
Abbamonte, Louis W., Capt., 29-15 Bell Blvd., Bayside.
Accardi, Vincent, Capt., 189 Weirfield St., Brooklyn.
Alden, Carlos C. Jr., Capt., 27 Ashland Ave., Buffalo.
Alderman, Henry R., Capt., Lewiston.
Allen, Herbert B., Capt., 216 Halstead Ave., Harrison.
Aliberto, John E. V., Capt., 58 Columbus Ave., Tuckahoe.
Alpert, Samuel, Capt., 620 Trinity Ave., Bronx.
Alterman, Robert H., Capt., 86 Georgia Ave., Long Beach.
Altman, Morris A., Capt., 5706 11th Ave., Brooklyn.
Amatrano, Vincent P., Major, 635 E. Forham Rd., New York.
Aprile, Ignatius J., Capt., 32-43 83d St., Jackson Heights, L. I.
Argue, John F., Capt., Lake St., Wilson.
Bachrach, Edgar H., Capt., 565 Manhattan Ave., New York.
Bakst, Hyman, Capt., 915 W. End Ave., New York.
Balser, Benjamin H., Lt. Col., 44 E. 67th St., New York.
Baronberg, Louis J., Capt., 1945 E. 18th St., Brooklyn.
Bashein, Gus, Major, 96-09 66th Ave., Forest Hills.
Battaglia, Richard S., 1st Lt., 323 E. 3d Ave., Roselle.
Bauer, Paul H., 1st Lt., Grasslands Hosp., Valahalla.
Baumann, Frank A. Jr., Major, 52 Schiller St., Binghamton.
Belden, Jerry J., Major, 329 High St., Watertown.
Bergnes, Manuel A., Major, 10 Bennett Ave., New York.
Berkowitz, Sidney B., 1st Lt., 1743 E. 4th St., Brooklyn.
Berlinghof, Clifton H., Lt. Col., 18 Avon Rd., Binghamton.
Berman, Leon G., Major, 125 Edgemont St., Syracuse.
Bertuglia, Sebastian, Capt., 131 Linden St., Brooklyn.
Bick, Malcolm W., Capt., 25 Minetta Lane, New York.
Biller, Samuel B., Major, 450 St. John's Pl., Brooklyn.
Bing, R. J., Capt., Arlington Ave. & W. 245th St., Riverdale.
Birchall, Robert, Capt., Port Washington.
Blankfield, Nathan, Major, 1506 Ocean Ave., Brooklyn.
Block, Harry H., Capt., 265 Quentin Rd., Brooklyn.
Bobeck, Charles J., Capt., 48 N. Main, Cannadiaqua.
Bolitho, Thomas B., Capt., Swenden-Walker Rd., Brockport.
Bloom, Bernard, Capt., 2153 E. 26th Ct., Brooklyn.
Bluestone, N. B., Major, Crompond Rd., Yorktown Heights.
Bochow, George A., Capt., 29 Pearl St., Mount Vernon.
Bock, Fred, Major, 88-32 150th St., Jamaica.
Bogue, Robert M., Capt., 140 Union Ave., Lynbrook.
Bopp, Ignatius M., Capt., 2838 Quinby Ave., New York.
Bosworth, Boardman M., Major, 915 Palmer Rd., Bronxville.
Botwin, Nathan, Major, 504 Grand St., New York.
Bratt, Murry A., Capt., 24 Monroe St., Honeoye Falls.
Bray, Harold P., Capt., 30-48 36th St., Long Island City.
Brezin, David, Capt., Mt. Sinai Hosp., New York.
Briggs, Bernard D., Major, 11 Main St., McGraw.
Brown, Francis D., Capt., Hobart.
Bruno, Michael J., Capt., 771 Bushwick Ave., Brooklyn.
Bryer, Morton S., Capt., Mt. Sinai Hosp., New York.
Buchanan, A. C. Jr., Capt., 115 Kingsbury Rd., Garden City, L. I.
Burke, John, Lt. Col., 2787 Main St., Buffalo.
Burns, John B., Major, 153 Chapin St., Binghampton.
Camardella, Ralph A., Lt. Col., 425 Eastern Parkway, Brooklyn.
Carr, Roland B., Capt., 1180 E. Ferry St., Buffalo.
Case, Wickham F., Capt., 359 Main St., Patchogue, L. I.
Cassara, Thomas, Major, Letchworth Village, Thiells.
Chakales, Harry J., Capt., 34 Plaza St., Brooklyn.
Chapman, David H., Major, 1929 E. Genesee St., Syracuse.
Charlton, Alex, Capt., 168th St. & Gerard Ave., Bronx.
Chiarulli, Eugene E., Major, 504 Prospect Ave., Syracuse.
Chillemi, Philip A., Capt., 146-08 Queens Ave., Flushing.
Cibelli, Ludwig J., Capt., 200 E. 18th St., Brooklyn.
Clement, David H., Capt., 135 Linwood Ave., Buffalo.
Cleveland, Mather, Col., Palisade Ave., Riverdale, New York.
Combes, J. de Daismes, Major, 6-34 150th, Whitestone.
Costigan, Thomas J., Major, 50-34 41st St., L. I. City.

PHYSICIANS SEPARATED FROM SERVICE

New York—Continued

Cove, Arthur M., Major, 395 Ocean Ave., Brooklyn.
Coxon, William F. Jr., Capt., 10 Bonnie Brae, Utica.
Cryst, John E., Capt., 2254 Main St., Buffalo.
Cullen, Milton L., Capt., 622 W. 168th St., New York.
Custer, Edward A., Major, 1341 Mt. Hope Ave., Rochester.
Dack, Simon, Capt., 174 W. 76th St., New York.
Dake, Charles M. Jr., Capt., Temperance House, Niagara Falls.
Dann, Sidney, Capt., 1700 E. 7th St., Brooklyn.
Davis, Ralph H., Lt. Col., Maple St., Addison.
Davis, William, Capt., 27 Park Pl., Great Neck.
Degenshein, George A., Capt., 1421 E. 7th St., Brooklyn.
Delalla, Emanuel, Lt. Col., 42 Brookline Dr., Utica.
de Rouville, William H., Capt., 190 Milner Ave., Albany.
deSiervo, Thomas G., Capt., 1330 Odell St., Bronx.
Dickinson, Meredith M., Capt., 270 Seamen Ave., New York.
Di Gregorio, N. J., Major, 1583 Bay Ridge Pkwy, Brooklyn.
Dimino, Michele S., Capt., 103-02 29th Ave. E., Elmhurst, L. I.
Doctrow, William M., Major, 1269 E. 10th St., Brooklyn.
Dooneief, Alfred S., Major, 175 Eastern Parkway, Brooklyn.
Dormont, Richard E., Capt., 123 E. 35th St., Brooklyn.
Dornberger, George R., Major, Troy.
Dorsey, John J., Capt., 321 E. 42d St., New York.
Doubilet, Henry, Capt., 25 E. 86th St., New York.
Douglas, Edward W., Major, 285 Oxford St., Rochester.
Douglass, Edwin H. Jr., Capt., 286 Liberty St., Newburgh.
Driggs, Marshall F., Capt., 50 W. 67th St., New York.
Durante, Dante V., Capt., 3200 Decatur Ave, Bronx.
Dwyer, Thomas A., Capt., 4541 DeLatield Ave., Bronx.
Earl, Alfred R., Major, Fayetteville.
Eggert, Edward T., Major, Knowlesville.
Eisenstater, Desider, Capt., 115 Logan St., Brooklyn.
Engster, Henry C., Capt., 186 2d Ave., N. Troy.
Errico, Frank J., Capt., 95 Washington Ave., Suffern.
Eustace, Charles G., Major, 320 East St., Buffalo.
Fabricant, Benjamin, Capt., 98-17 Queens Blvd., Forest Hills.
Fausel, Everett G., Major, 88-15 192d St., Hollis.
Feichtner, Leo V., Major, 8 Sunset Dr., Croton-on-Hudson.
Feig, Milton, 1st Lt., 66 Park Ave., Manhasset.
Feit, Leonard, Capt., Gouverneur Hosp., New York.
Feuer, Abe L., Capt., 984 E. 178th St., Bronx.
Fiandaca, Patsy M. Jr., Capt., 60 Lincoln Ave., Poughkeepsie.
Field, William H., Col., 67 Willow St., Brooklyn.
Finegola, John, Capt., 655 Main, Niagara Falls.
Fink, Benjamin, Capt., 242 Vernon Ave., Brooklyn.
Finkelstein, Nathan E., Capt., 194 Grafton St., Brooklyn.
Finn, Joseph W., Capt., 628 59th St., Brooklyn.
Fishman, Benjamin S., Capt., 310 E. 75th St., New York.
Fitzgerald, Gerald P., Capt., 168 N. Allen St., Albany.
Flynn, John J., Capt., 8302 Ridge Blvd., Brooklyn.
Franco, Saverio C., Lt. Col., 200 Clinton St., Brooklyn.
Frank, Cecil A., Major, 698 West End Ave., New York.
Frank, Norris H., Major, Pine St., South Dayton.
Friedenberg, Zachary B., Major, 601 W. 190th St., New York.
Friedenthal, Meyer, Capt., 1150 Garrison Ave., New York.
Friedman, Joseph, Capt., 57 Midwood St., Brooklyn.
Friedman, Sol, Capt., 1770 Davidson Ave., Bronx.
Gaeta, Louis E., Capt., 3758 93d St., Jackson Heights, L. I.
Gagnier, Gerard E., Capt., Churubusco.
Gainor, John E., Capt., 3 Groesbeck Pl., Elsmere, Albany Co.
Galiani, Joshua L., Capt., 2078 Prospect Ave., Bronx.
Garber, Jacob S., Capt., 89-24 146th St., Jamaica, L. I.
Gaston, Sawnie R., Capt., 40 Monroe St., New York.
Gaydos, Albert L., Capt., 153 Eagle St., Brooklyn.
George, John D. Jr., Capt., Verona, Oneida County.
Gerlin, George J., Capt., 361 Irving Ave., Brooklyn.
Gershwin, Benjamin S., Capt., 322 Malboro Rd., Brooklyn.
Gerstein, Jerome W., Capt., 1284 Stebbins Ave., Bronx.
Gerstner, Martin L., Major, 27 Admiral Rd., Buffalo.
Gesell, Eric G., Capt., 48-11 Bell Blvd., Bayside, L. I.
Geshwind, Morris H., Capt., 676 Montgomery St., Brooklyn.
Getman, Edson E., Lt. Col., 44 E. 57th St., New York.
Gevjan, Armen H., 1st Lt., 1804 Paulding Ave., New York.
Gibbs, Donald R., Capt., 110 N. Main St., Cortland.
Giddings, Emanuel, Col., 26th St. & 1st Ave., New York.
Giorgini, Gino L., Capt., Great Neck Rd., Copiague, L. I.
Gittell, George, Capt., 1775 Clay Ave., New York.
Gold, Irving B., Capt., 330 W. 30th St., New York.
Golding, George T., Capt., 7301 19th Ave., Brooklyn.
Goldman, Louis, Capt., 1060 Sherman Ave., New York.
Goldman, Morris, Capt., 111-54 Farmers Blvd., St. Albans.
Goldstein, Moe, Capt., 108-25 72d Ave., Forest Hills, L. I.
Good, Conrad E., Capt., 20 Modelane, Brighton.
Goodyear, Emils S., Major, 61 Maiden Lane, Kingston.

New York—Continued

Gottlieb, Moses L., Major, 955 Walton Ave., New York.
Graczyk, Stephen A., Major, 54 Park Blvd., Lancaster.
Graef, Irving P., Major, 1109 Madison Ave., New York.
Grayson, Morris, Major, 97-10 103d, Ozone Park, L. I.
Greenfield, Irving, Capt., 115 Ocean Ave., Brooklyn.
Gromet, Maurice T., Capt., 125 Ocean Ave., Brooklyn.
Grosselfinger, Harold W., Capt., 297 Dartmouth St., Rochester.
Gutelius, Stanley K., Capt., 388 Magee Ave., Rochester.
Haber, Mordecai, Capt., 1822 Mohegan Ave., New York.
Haight, Julius R., Major, Harlem Valley State Hosp., Wingdale.
Hamilton, Charles K., Capt., 315 E. 68th St., New York.
Hardy, Edward A., Capt., 346 Corlies Ave., Pelham.
Harris, Harold M., Capt., 118 Ashland Ave., Buffalo.
Harrity, James G., Capt., 2360 S. Park Ave., Buffalo.
Harrow, D. G., Capt., 342 Ft. Washington Ave., New York.
Hartig, S. J., Capt., 459 Pelhamdale Ave., Pelham Manor.
Heiligman, S. A., Capt., % Lipshitz, 990 President, Brooklyn.
Hellen, Sidney L., Capt., 1133 Morrison Ave., Bronx.
Himmelstein, Aaron, Capt., 235 E. 22d St., New York.
Hirschfeld, Walter L., 1st Lt., 128 Hamilton St., Olean.
Hoak, Frank C. Jr., Major, 235 Sterling Ave., Buffalo.
Horn, Leonard, Capt., 306 Laburnum Crescent, Rochester.
Huber, Frank, Lt. Col., 16 Sherman Ave., White Plains.
Hughes, Robert J., Major, 572 Sterling Pl., Brooklyn.
Hutchings, Charles W., Lt. Col., Marcy State Hosp., Marcy.
Ilardi, Onofrio, Capt., 103-19 103d Ave., Ozone Park.
Itzkowitz, Louis E., Major, 5303 12th Ave., Brooklyn.
Jackson, Abraham, Major, 24-05 28th St., Astoria, L. I.
Jaffe, Irwin, Capt., 2108 Avenue L, Brooklyn.
James, William M., Capt., 669 Tinton Ave., Bronx.
Jenkins, Charles R., Major, 509 Bainbridge St., Brooklyn.
Johnson, Frank C., 1st Lt., 244 Putnam Ave., Brooklyn.
Johnson, Ted, Lt. Col., 418 Central Park, New York.
Jones, Thomas B., Col., 11 S. Goodman St., Rochester.
Kaplan, Leo, Capt., 870 Elsmere Pl., Bronx.
Kalow, Irving, Capt., 2230 Grand Concourse, New York.
Katz, Alexander A., Capt., 1050A President St., Brooklyn.
Katz, Simon, Capt., 959 Carroll St., Brooklyn.
Kaufman, Robert E., Capt., 50 E. 78th St., New York.
Kenwell, Henry N., Lt. Col., 43 Summit Ave., Buffalo.
Kessler, Franklin L., Capt., 1280 Maple Ave., Peekskill.
Kidder, James H., Lt. Col., 5031 Fieldstone Rd., New York.
Kilpatrick, Oswald A., Lt. Col., Willard State Hosp., Willard.
Kirban, Harry N., Capt., Grasslands Hosp., Valhalla.
Kirsner, Morris H., Major, 694 Yonkers Ave., Yonkers.
Klein, Lawrence A., Capt., 1524 Culver Rd., Rochester.
Kleinman, Egon, Capt., 127 W. 82d St., New York.
Klingenstein, Percy, Col., 1070 Park Ave., New York.
Knapp, Irving J., Capt., 23 Clinton Ave., Warwick.
Knoll, Alfred F., Major, 617 W. 168th St., New York.
Kohn, Anthony, Capt., Vet. Adm., Northport, L. I.
Koschir, Frank J., Capt., 440 Harman St., Brooklyn.
Krauss, George G., Capt., 4104 76th St., Jackson Hgts., L. I.
Kreusser, Edward J., Capt., 7022 Ridge Blvd., Brooklyn.
Krugman, Benjamin M., Capt., 66 Sussex St., Port Jervis.
Krumdieck, Newton, Capt., 525 E. 68th St., New York.
Kurek, Leon S., Lt. Col., 761 Fillmore Ave., Buffalo.
Kutner, Charles, Capt., 1005 S. 5th St., Camden.
Lampert, Norman J., Capt., 86 Bulwer Pl., Brooklyn.
Lande, Herman, Col., 25 E. 67th St., New York.
Lansman, Henry H., Capt., 189-02A 35th Ave., Flushing.
Learn, Marshall L., Major, 59 Buffalo St., Hamburg.
Lefkovits, Aaron M., Major, 1340 St. Lawrence Ave., Bronx.
Lepler, Morton M., 1st Lt., 639 W. 204th St., New York.
Leve, Howard B., Capt., 43-15 46th St., Sunnyside, L. I.
Levin, Robert R., Capt., 752 W. End Ave., New York.
Levine, Julius, Major, Swan Lake.
Levine, Louis, Major, 5903 15th Ave., Brooklyn.
Lewis, Benjamin, Major, 340 Riverside Dr., New York.
Linn, Louis, Capt., 3070 Hull Ave., Bronx.
Lipton, Harold, Major, 1819 Avenue L, Brooklyn.
Localio, Silvio A., Capt., N. Y. Post Grad. Hosp., New York.
Locasto, Camillo B., Capt., 276 Linden St., Brooklyn.
Loizeaux, Marion C., Major, 218 E. 36th St., New York.
Lombardo, Ernest A., 1st Lt., 428 Park Pl., Brooklyn.
Lubitz, Benjamin, Col., 1344 50th St., Brooklyn.
Lucey, John K., Major, 390 Van Duzer St., Stapleton, S. I.
Lyons, John J. A., Major, 15 Joelson Ct., Albany.
McAteer, Daniel A., Capt., 3123 Avenue J, Brooklyn.
McConnell, W. J., Col., Box 26, Madison Sq. P. O., New York.
McGan, Harold P., Lt. Col., 302 Western Ave., Albany.
McGowan, John E., Capt., Rockland State Hosp., Orangeburg.

A NATIONAL HEALTH PROGRAM

Message to Congress on a National Health Program from President Harry S. Truman

To the Congress of the United States:

In my message to the Congress of Sept. 6, 1945 there were enumerated in a proposed economic bill of rights certain rights which ought to be assured to every American citizen.

One of them was "the right to adequate medical care and the opportunity to achieve and enjoy good health." Another was the "right to adequate protection from the economic fears of . . . sickness. . . ."

Millions of our citizens do not now have a full measure of opportunity to achieve and enjoy good health. Millions do not now have protection or security against the economic effects of sickness. The time has arrived for action to help them attain that opportunity and that protection.

The people of the United States received a shock when the medical examinations conducted by the Selective Service System revealed the widespread physical and mental incapacity among the young people of our nation. We had had prior warnings from eminent medical authorities and from investigating committees. The statistics of the last war had shown the same condition. But the Selective Service System has brought it forcibly to our attention recently in terms which all of us can understand.

As of April 1, 1945 nearly 5,000,000 male registrants between the ages of 18 and 37 had been examined and classified as unfit for military service. The number of those rejected for military service was about 30 per cent of all those examined. The percentage of rejection was lower in the younger age groups and higher in the higher age groups, reaching as high as 49 per cent for registrants between the ages of 34 and 37. In addition, after actual induction, about a million and a half men had to be discharged from the Army and Navy for physical or mental disability, exclusive of wounds; and an equal number had to be treated in the armed forces for diseases or defects which existed before induction.

Among the young women who applied for admission to the Women's Army Corps there was similar disability. Over one third of those examined were rejected for physical or mental reasons.

These men and women who were rejected for military service are not necessarily incapable of civilian work. It is plain, however, that they have illnesses and defects that handicap them, reduce their working capacity or shorten their lives.

It is not so important to search the past in order to fix the blame for these conditions. It is more important to resolve now that no American child shall come to adult life with diseases or defects which can be prevented or corrected at an early age.

Medicine has made great strides in this generation, especially during the last four years. We owe much to the skill and devotion of the medical profession. In spite of great scientific progress, however, each year we lose many more persons from preventable and premature deaths than we lost in battle or from war injuries during the entire war.

We are proud of past reductions in our death rates. But these reductions have come principally from public health and other community services. We have been less effective in making available to all of our people the benefits of medical progress in the care and treatment of individuals.

In the past, the benefits of modern medical science have not been enjoyed by our citizens with any degree of equality. Nor are they today. Nor will they be in the future unless government is bold enough to do something about it.

People with low or moderate incomes do not get the same medical attention as those with high incomes. The poor have more sickness, but they get less medical care. People who live in rural areas do not get the same amount or quality of medical attention as those who live in our cities. Our new economic bill of rights should mean health security for all, regardless of residence, station or race everywhere in the United States. We should resolve now that the health of this nation is a national concern; that financial barriers in the way of attaining health shall be removed; that the health of all its citizens deserves the help of all the nation.

There are five basic problems which we must attack vigorously if we would reach the health objectives of our economic bill of rights:

Distribution

1. The first has to do with the number and distribution of doctors and hospitals. One of the most important requirements for adequate health service is professional personnel—doctors, dentists, public health and hospital administrators, nurses and other experts. The United States has been fortunate with respect to physicians. In proportion to population it has more than any large country in the world, and they are well trained for their calling. It is not enough, however, that we have them in sufficient numbers. They should be located where their services are needed. In this respect we are not so fortunate. The distribution of physicians in the United States has been grossly uneven and unsatisfactory. Some communities have had enough or even too many; others have had too few. Year by year the number in our rural areas has been diminishing. Indeed, in 1940 there were thirty-one counties in the United States, each with more than a thousand inhabitants, in which there was not a single practicing physician. The situation with respect to dentists was even worse.

One important reason for this disparity is that in some communities there are no adequate facilities for the practice of medicine. Another reason, closely allied with the first, is that the earning capacity of the people in some communities makes it difficult if not impossible for doctors who practice there to make a living.

The demobilization of 60,000 doctors and of the tens of thousands of other professional personnel in the armed forces is now proceeding on a large scale. Unfortunately, unless we act rapidly, we may expect to see them concentrate in the places with greater financial resources and avoid other places, making the inequalities even greater than before the war. Demobilized doctors cannot be assigned. They must be attracted. In order to be attracted, they must be able to see ahead of them professional opportunities and economic assurances.

Inequalities in the distribution of medical personnel are matched by inequalities in hospitals and other health facilities. Moreover, there are just too few hospitals, clinics and health centers to take proper care of the people of the United States.

About 1,200 counties, 40 per cent of the total in the country, with some 15,000,000 people, have either no local hospital or none that meets even the minimum standards of national professional associations. The deficiencies are especially severe in rural and semirural areas and in those cities where changes in population have placed great strains on community facilities. I want to emphasize, however, that the basic problem in this field cannot be solved merely by building facilities. They have to be staffed; and the communities have to be able to pay for the services. Otherwise the new facilities will be little used.

Maternal and Child Care

2. The second basic problem is the need for development of public health services and maternal and child care. The Congress can be justifiably proud of its share in making recent accomplishments possible. Public health and maternal and child health programs already have made important contributions to national health. But large needs remain. Great areas of our country are still without these services. This is especially true among our rural areas; but it is true also in far too many urban communities.

Although local public health departments are now maintained by some 18,000 counties and other local units, many of these have only skeleton organizations, and approximately 40,000,000 citizens of the United States still live in communities lacking full time local public health service. At the recent rate of progress in developing such service, it would take more than a hundred years to cover the whole nation.

If we agree that the national health must be improved, our cities, towns and farming communities must be made healthful places in which to live through provision of safe water systems, sewage disposal plants and sanitary facilities. Our streams and rivers must be safeguarded against pollution. In addition to building a sanitary environment for ourselves and for our children, we must provide those services which prevent disease and promote health.

Services for expectant mothers and for infants, care of crippled or otherwise physically handicapped children and inoculation for the prevention of communicable diseases are accepted public health functions. So too are many kinds of personal services such as the diagnosis and treatment of widespread infections like tuberculosis and venereal disease. A large part of the population today lacks many or all of these services.

Our success in the traditional public health sphere is made plain by the conquest over many communicable diseases. Typhoid fever, smallpox and diphtheria—diseases for which there are effective controls—have become comparatively rare. We must make the same gains in reducing our maternal and infant mortality, in controlling tuberculosis, venereal disease, malaria and other major threats to life and health. We are only beginning to realize our potentialities in achieving physical well-being for all our people.

Research and Education

3. The third basic problem concerns medical research and professional education. We have long recognized that we cannot be content with what is already known about health or disease. We must learn and understand more about health and how to prevent and cure disease.

Research—well directed and continuously supported—can do much to develop ways to reduce those diseases of body and mind which now cause most sickness, disability and premature death—diseases of the heart, kidneys and arteries, rheumatism, cancer, diseases of childbirth, infancy and childhood, respiratory diseases and tuberculosis. And research can do much toward teaching us how to keep well and how to prolong healthy human life.

Cancer is among the leading causes of death. It is responsible for over 160,000 recorded deaths a year and should receive special attention. Though we already have the National Cancer Institute of the Public Health Service, we need still more coordinated research on the cause, prevention and cure of this disease. We need more financial support for research and to establish special clinics and hospitals for diagnosis and treatment of the disease, especially in its early stages. We need to train more physicians for the highly specialized services so essential for effective control of cancer.

There is also special need for research on mental diseases and abnormalities. We have done pitifully little about mental illnesses. Accurate statistics are lacking, but there is no doubt that there are at least two million persons in the United States who are mentally ill and that as many as ten million will probably need hospitalization for mental illness for some period in the course of their lifetime. A great many of these persons would be helped by proper care. Mental cases occupy more than one half of the hospital beds at a cost of about 500 million dollars per year, practically all of it coming out of taxpayers' money. Each year there are 125,000 new mental cases admitted to institutions. We need more mental disease hospitals, more outpatient clinics. We need more services for early diagnosis, and especially we need much more research to learn how to prevent mental breakdown. Also we must have many more trained and qualified doctors in this field.

It is clear that we have not done enough in peacetime for medical research and education in view of our enormous resources and our national interest in health progress. The money invested in research pays enormous dividends. If any one doubts this, let him think of penicillin, plasma, DDT powder and new rehabilitation technics.

Cost of Medical Care

4. The fourth problem has to do with the high cost of individual medical care. The principal reason why people do not receive the care they need is that they cannot afford to pay for it on an individual basis at the time they need it. This is true not only for needy persons. It is also true for a large proportion of normally self-supporting persons.

In the aggregate all health services—from public health agencies, physicians, hospitals, dentists, nurses and laboratories—absorb only about 4 per cent of the national income. We can afford to spend more for health. But 4 per cent is only an average. It is cold comfort in individual cases. Individual families pay their individual costs and not average costs. They may be hit by sickness that calls for many times the average cost—in extreme cases for more than their annual income. When this happens they may come face to face with economic disaster. Many families, fearful of expense, delay calling the doctor long beyond the time when medical care would do the most good. For some persons with very low income or no income at all we now use taxpayers' money in the form of free services, free clinics and public hospitals. Tax-supported, free medical care for needy persons, however, is insufficient in most of our cities and in nearly all of our rural areas. This deficiency cannot be met by private charity or the kindness of individual physicians. Each of us knows doctors who work through endless days and nights, never expecting to be paid for their services because many of their patients are unable to pay. Often the physician spends not only his time and effort but even part of the fees he has collected from patients able to pay, in order to buy medical supplies for those who cannot afford them. I am sure that there are thousands of such physicians throughout our country. They cannot, and should not, be expected to carry so heavy a load.

Compensation

5. The fifth problem has to do with loss of earnings when sickness strikes. Sickness not only brings doctor bills; it also cuts off income. On an average day there are about 7 million persons so disabled by sickness or injury that they cannot go about their usual tasks. Of these, about 3¼ millions are persons who, if they were not disabled, would be working or seeking employment. More than one half of these disabled workers have already been disabled for six months; many of them will continue to be disabled for years, and some for the remainder of their lives. Every year four or five hundred million working days are lost from productive employment because of illness and accident among those working or looking for work—about forty times the number of days lost because of strikes on the average during the ten years before the war. About nine tenths of this enormous loss is due to illness and accident that is not directly connected with employment and is therefore not covered by workmen's compensation laws.

These, then, are the five important problems which must be solved if we hope to attain our objective of adequate medical care, good health and protection from the economic fears of sickness and disability.

To meet these problems, I recommend that the Congress adopt a comprehensive and modern health program for the nation, consisting of five major parts — each of which contributes to all the others.

Construction

First: Construction of hospitals and related facilities. The federal government should provide financial and other assistance for the construction of needed hospitals, health centers and other medical, health and rehabilitation facilities. With the help of federal funds it should be possible to meet deficiencies in hospital and health facilities so that modern services — for both prevention and cure — can be accessible to all the people. Federal financial aid should be available not only to build new facilities where needed but also to enlarge or modernize those we now have.

In carrying out this program there should be a clear division of responsibilities between the states and the federal government. The states, localities and the federal government should share in the financial responsibilities. The federal government should not construct or operate these hospitals. It should, however, lay down minimum national standards for construction and operation and should make sure that federal funds are allocated to those areas and projects where federal aid is needed most. In approving state plans and individual projects, and in fixing the national standards, the federal agency should have the help of a strictly advisory body that includes both public and professional members. Adequate emphasis should be given to facilities that are particularly useful for prevention of diseases—mental as well as physical—and to the coordination of various kinds of facilities. It should be possible to go a long way toward knitting together facilities for prevention with facilities for cure, the large hospitals of medical centers with the smaller institutions of surrounding areas, the facilities for the civilian population with the facilities for veterans. The general policy of federal-state partnership which has done so much to provide the magnificent highways of the United States can be adapted to the construction of hospitals in the communities which need them.

Expansion of Health Services

Second: Expansion of public health, maternal and child health services. Our programs for public health and related services should be enlarged and strengthened. The present federal-state cooperative health programs deal with general public health work, tuberculosis and venereal disease control, maternal and child health services, and services for crippled children. These programs were especially developed in the ten years before the war and have been extended in some areas during the war. They have already made important contributions to national health, but they have not yet reached a large proportion of our rural areas, and in many cities they are only partially developed.

No area in the nation should continue to be without the services of a full time health officer and other essential personnel. No area should be without essential public health services or sanitation facilities. No area should be without community health services such as maternal and child health care. Hospitals, clinics and health centers must be built to meet the needs of the total population and must make adequate provision for the safe birth of every baby, and for the health protection of infants and children.

Present laws relating to general public health, and to maternal and child health, have built a solid foundation of federal cooperation with the states in administering community health services. The emergency maternity and infant care program for the wives and infants of servicemen—a great wartime service authorized by the Congress—has materially increased the experience of every state health agency and has provided much needed care. So too have other wartime programs such as venereal disease control, industrial hygiene, malaria control, tuberculosis control and other services offered in war essential communities. The federal government should cooperate by more generous grants to the states than are provided under present laws for public health services and for maternal and child health care. The program should continue to be partly financed by the states themselves and should be administered by the states. Federal grants should be in proportion to state and local expenditures and should also vary in accordance with the financial ability of the respective states.

The health of American children, like their education, should be recognized as a definite public responsibility. In the conquest of many diseases prevention is even more important than cure. A well rounded national health program should therefore include systematic and widespread health and physical education and examinations, beginning with the youngest children and extending into community organizations. Medical and dental examinations of school children are now inadequate. A preventive health program, to be successful, must discover defects as early as possible. We should therefore see to it that our health programs are pushed most vigorously with the youngest section of the population. Of course, federal aid for community health services—for general public health and for mothers and children—should complement and not duplicate prepaid medical services for individuals, proposed by the fourth recommendation of this message.

Medical Education and Research

Third: Medical education and research. The federal government should undertake a broad program to strengthen professional education in medical and related fields and to encourage and support medical research. Professional education should be strengthened where necessary through federal grants-in-aid to public and to nonprofit private institutions. Medical research also should be encouraged and supported in the federal agencies and by grants-in-aid to public and nonprofit private agencies.

In my message to the Congress of Sept. 6, 1945 I made various recommendations for a general federal research program. Medical research, dealing with the broad fields of physical and mental illnesses, should be made effective in part through that general program and in part through specific provisions within the scope of a national health program. Federal aid to promote and support research in medicine, public health and allied fields is an essential part of a general research program to be administered by a central federal research agency. Federal aid for medical research and education is also an essential part of any national health program if it is to meet its responsibilities for high grade medical services and for continuing progress. Coordination of the two programs is obviously necessary to assure efficient use of federal funds. Legislation covering medical research in a national health program should provide for such coordination.

Medical Costs

Fourth: Prepayment of medical costs. Every one should have ready access to all necessary medical, hospital and related services. I recommend solving the basic problem by distributing the costs through expansion of our existing compulsory social insurance system. This is not socialized medicine. Every one who carries fire insurance knows how the law of averages is made to work so as to spread the risk and to benefit the insured who actually suffers the loss. If, instead of the costs of sickness being paid only by those who get sick, all the people—sick and well—were required to pay premiums into an insurance fund, the pool of funds thus created would enable all who do fall sick to be adequately served without overburdening any one. That is the principle on which all forms of insurance are based.

During the past fifteen years, hospital insurance plans have taught many Americans this magic of averages. Voluntary health insurance plans have been expanding during recent years; but their rate of growth does not justify the belief that they will meet more than a fraction of our people's needs. Only about 3 per cent or 4 per cent of our population now have insurance providing comprehensive medical care. A system of required prepayment would not only spread the costs of medical care, it would also prevent much serious disease. Since medical bills would be paid by the insurance fund, doctors would more often be consulted when the first signs of disease occur instead of when the disease has become serious. Modern hospital, specialist and laboratory services, as needed, would also become available to all and would improve the quality and adequacy of care. Prepayment of medical care would go a long way toward furnishing insurance against disease itself as well as against medical bills. Such a system of prepayment should cover medical, hospital, nursing and laboratory services. It should also cover dental care as fully and for as many of the population as the available professional personnel and the financial resources of the system permit.

The ability of our people to pay for adequate medical care will be increased if, while they are well, they pay regularly into a common health fund instead of paying sporadically and unevenly when they are sick. This health fund should be built up nationally in order to establish the broadest and most stable basis for spreading the costs of illness and to assure adequate financial support for doctors and hospitals everywhere. If we were to rely on state by state action only, many years would elapse before we had any general coverage. Meanwhile health

service would continue to be grossly uneven, and disease would continue to cross state boundary lines.

Medical services are personal. Therefore the nationwide system must be highly decentralized in administration. The local administrative unit must be the keystone of the system so as to provide for local services and adaptation to local needs and conditions. Locally as well as nationally, policy and administration should be guided by advisory committees in which the public and the medical professions are represented.

Subject to national standards, methods and rates of paying doctors and hospitals should be adjusted locally. All such rates for doctors should be adequate and should be appropriately adjusted upward for those who are qualified specialists. People should remain free to choose their own physicians and hospitals. The removal of financial barriers between patient and doctor would enlarge the present freedom of choice. The legal requirement on the population to contribute involves no compulsion over the doctor's freedom to decide what services his patient needs. People will remain free to obtain and pay for medical service outside the health insurance system if they desire, even though they are members of the system, just as they are free to send their children to private instead of to public schools, although they must pay taxes for public schools. Likewise physicians should remain free to accept or reject patients. They must be allowed to decide for themselves whether they wish to participate in the health insurance system full time, part time or not at all. A physician may have some patients who are in the system and some who are not. Physicians must be permitted to be represented through organizations of their own choosing and to decide whether to carry on in individual practice or to join with other doctors in group practice in hospitals or in clinics.

Our voluntary hospitals and our city, county and state general hospitals, in the same way, must be free to participate in the system to whatever extent they wish. In any case they must continue to retain their administrative independence.

Voluntary Organizations

Voluntary organizations which provide health services that meet reasonable standards of quality should be entitled to furnish services under the insurance system and to be reimbursed for them. Voluntary cooperative organizations concerned with paying doctors, hospitals or others for health services, but not providing services directly, should be entitled to participate if they can contribute to the efficiency and economy of the system.

None of this is really new. The American people are the most insurance minded people in the world. They will not be frightened off from health insurance because some people have misnamed it "socialized medicine."

I repeat—what I am recommending is not socialized medicine. Socialized medicine means that all doctors work as employees of government. The American people want no such system. No such system is here proposed. Under the plan I suggest, our people would continue to get medical and hospital services just as they do now—on the basis of their own voluntary decisions and choices. Our doctors and hospitals would continue to deal with disease with the same professional freedom as now. There would, however, be this all important difference: Whether or not patients get the services they need would not depend on how much they can afford to pay at the time.

I am in favor of the broadest possible coverage for this insurance system. I believe that all persons who work for a living and their dependents should be covered under such an insurance plan. This would include wage and salary earners, those in business for themselves, professional persons, farmers, agricultural labor, domestic employees, government employees and employees of nonprofit institutions and their families. In addition, needy persons and other groups should be covered through appropriate premiums paid for them by public agencies. Increased federal funds should also be made available by the Congress under the public assistance programs to reimburse the states for part of such premiums, as well as for direct expenditures made by the states in paying for medical services provided by doctors, hospitals and other agencies to needy persons.

Premiums for present social insurance benefits are calculated on the first $3,000 of earnings in a year. It might be well to have all such premiums, including those for health, calculated on a somewhat higher amount, such as $3,600. A broad program of prepayment for medical care would need total amounts approximately equal to 4 per cent of such earnings. The people of the United States have been spending, on the average, nearly this percentage of their incomes for sickness care. How much of the total fund should come from the insurance premiums and how much from general revenues is a matter for the Congress to decide. The plan which I have suggested would be sufficient to pay most doctors more than the best they have received in peacetime years. The payments of the doctors' bills would be guaranteed, and the doctors would be spared the annoyance and uncertainty of collecting fees from individual patients. The same assurance would apply to hospitals, dentists and nurses for the services they render. Federal aid in the construction of hospitals will be futile unless there is current purchasing power so that people can use these hospitals. Doctors cannot be drawn to sections which need them without some assurance that they can make a living. Only a nationwide spreading of sickness costs can supply such sections with sure and sufficient purchasing power to maintain enough physicians and hospitals. We are a rich nation and can afford many things. But ill health which can be prevented or cured is one thing we cannot afford.

Comprehensive Health Program

Fifth: Protection against loss of wages from sickness and disability. What I have discussed heretofore has been a program for improving and spreading the health services and facilities of the nation and providing an efficient and less burdensome system of paying for them. But no matter what we do, sickness will of course come to many. Sickness brings with it loss of wages. Therefore, as a fifth element of a comprehensive health program, the workers of the nation and their families should be protected against loss of earnings because of illness. A comprehensive health program must include the payment of benefits to replace at least part of the earnings that are lost during the period of sickness and long term disability. This protection can be readily and conveniently provided through expansion of our present social insurance system, with appropriate adjustment of premiums.

Insurance against loss of wages from sickness and disability deals with cash benefits rather than with services. It has to be coordinated with the other cash benefits under existing social insurance systems. Such coordination should be effected when other social security measures are reexamined. I shall bring this subject again to the attention of the Congress in a separate message on social security.

I strongly urge that the Congress give careful consideration to this program of health legislation now.

Many millions of our veterans, accustomed in the armed forces to the best of medical and hospital care, will no longer be eligible for such care as a matter of right except for their service connected disabilities. They deserve continued adequate and comprehensive health service. And their dependents deserve it too.

By preventing illness, by assuring access to needed community and personal health services, by promoting medical research and by protecting our people against the loss caused by sickness we shall strengthen our national health, our national defense and our economic productivity. We shall increase the professional and economic opportunities of our physicians, dentists and nurses. We shall increase the effectiveness of our hospitals and public health agencies. We shall bring new security to our people.

We need to do this especially at this time because of the return to civilian life of many doctors, dentists and nurses, particularly young men and women.

Appreciation of modern achievements in medicine and public health has created widespread demand that they be fully applied and universally available. By meeting that demand we shall strengthen the nation to meet future economic and social problems; and we shall make a most important contribution toward freedom from want in our land.

ORGANIZATION SECTION

Washington Letter

(From a Special Correspondent)

Nov. 19, 1945.

Army Doctors May Be Released on 70 Points by December 15

The present score for the release of doctors by the Army is 80 points, but this may be reduced to 70 points by December 15. The Navy will drop its release score for doctors from 53 to 51 points on January 1. Navy dentists are released on the same scores as Navy line officers, who since November 1 have won discharge on 46 points. Surg. Gen. Norman T. Kirk reveals that he is pressing the War Department for speedier release of doctors and dentists. He is endeavoring to learn the minimum required overseas. General Kirk's office is also considering a recommendation that the Army cut its minimum discharge score to 10 points. At present the Army releases medical officers on 80 points or if they are 48 or had entered service before Pearl Harbor. Originally the Army had 45,000 medical men, and it expects to release 17,000 doctors and dentists by January 1. The rate of release is said to be faster than that for enlisted men. The Navy reveals that it is demobilizing three doctors to every thousand men, and, with 4,700 more doctors to be eligible soon, there are expected to be "few if any" doctors besides regular Navy doctors in service.

Senator Sheridan Downey, Democrat of California, endeavoring to learn how many doctors the Army is keeping in uniform, has asked the Surgeon General three questions: 1. What was the total troop enrolment and total number of doctors as of the first of every month from January 1945 through November? 2. Will you supply a list of all hospitals in the United States under the Surgeon General's command, together with the number of patients and doctors at each? 3. What is the total number of doctors assigned to the Veterans Administration?

"Almost a Million" Medical Examinations Given by Army

Two thousand service doctors and dentists in United States separation centers gave physical examinations to 572,119 outgoing Army men and women during the first twenty-four days of October. Additional service personnel received similar examinations from other doctors and dentists at other points, boosting the total number of examinations in the period to "almost a million." The physical examinations for discharge take about an hour. There are eight medical men available for each examination, the dischargee going to a dentist, an eye, ear, nose and throat specialist, an orthopedic surgeon for limb scrutiny, an internist for examination of the vital organs, a surgeon for study of scars, wounds or injuries, a laboratory man, and then a final check by an overall examiner, who may send the individual to a neuropsychiatrist for nerve tests. The Army revealed that medical school graduates are being commissioned and assigned as soon as they complete their internship. Most new medical men have been in the Army Student Training Program and are subject to immediate military assignment for the usual "duration and six months." All are assigned except those physically disqualified, the latter being reported as "relatively negligible."

Veterans Administration to Use AAF Hospital Planes

Army Air Forces hospital planes will be used by the Veterans Administration to fly patients from outlying hospitals to clinical centers. Previously patients have been moved by railroad, and long distances, particularly in Western areas, have been a serious handicap, but General Hawley, Veterans Acting Surgeon General, formerly Army chief surgeon in Europe, knows how air ambulances saved thousands of lives. Cooperation is expected from the AAF, which wants to keep a nominal number of hospital planes in operation. The agency will probably buy at least two hospital pullmans from surplus property stocks.

Further Changes in Veterans Administration

General Hawley is making an effort to use as many disabled veterans as possible in rehabilitation, prosthetics and other lines. The agency already has 123 disabled women veterans in training for its vocational rehabilitation program. The Nursing Advisory Council to the Administrator of Veterans Affairs has elected Ruth Sleeper, R.N., assistant principal of the Massachusetts General Hospital, Boston, as its chairman and has recommended to General Hawley (1) the establishment of a nurse corps in the Veterans Administration, (2) continuation of the development of in-service training, (3) postgraduate educational programs for Veterans Administration nurses and (4) appointment of a nursing advisory committee to the nurse representative in the thirteen Veteran Administration branch offices.

General Omar N. Bradley, Veterans Administrator, has announced changes in agency terminology. He has banned the use of the word "facility" to describe veterans' hospitals, in the belief that a hospital is a hospital. A domiciliary facility will be known as a Veterans Administration home. A hospital or home to which a regional office is attached will be called a Veterans Administration center instead of a "combined facility," as it has been known up to now.

Army Tuberculosis Expert Lent to Veterans Administration

To assure a superior standard of care for tuberculous veterans, Col. Esmond Ray Long of Philadelphia, described as Army's number 1 man on tuberculosis, has been lent to the Veterans Administration, on request of Major Gen. Paul R. Hawley, acting surgeon general. Colonel Long, chief consultant on tuberculosis to the Surgeon General of the Army, explains that the war did not develop any new therapies in the treatment of tuberculosis. The Army has depended on the familiar methods of "collapse therapy," including pneumothorax treatment. He said that the incidence of tuberculosis in this war is only one tenth of first world war figures. In this war it was 1.2 per thousand men annually as against 12 cases per thousand men annually in the first world war. Colonel Long only recently returned from Germany, where he was attached to the Office of Military Government.

Hospitals Get First Call on Penicillin

Because of a three month production slump, the War Production Board moved to give hospitals first call on all production. There is also opinion in favor of restoring export controls to check the amount leaving the country. An emergency directive will remain in force until production increases. Production dropped from 688,890,000 billion units in July to 568,370,000 billion units in September. It started up again in October, reaching 612,470,000 billion units. Estimated production is 657,000,000 billion units in November and 657,960,000 in December.

Army Reveals Half of Personnel Inoculated Against Influenza

About half of the U. S. Army has received inoculations against influenza and the rest will get them soon. The first recipients in this country were the staff of Walter Reed Hospital, with Major Gen. Norman T. Kirk, Army Surgeon General, and Major Gen. George F. Lull, Deputy Surgeon General, among those taking their shots. The program is expected to be completed in a few weeks. All overseas men have received their inoculation.

Shortage of Penicillin as a Result of Public Demand

Public demand for penicillin in ointment or lozenge form is blamed for a nationwide shortage of the drug reported by the Food and Drug Administration. Dr. Henry Welch, chief of the penicillin division of the Federal Drug Administration, said that the shortage will be overcome in sixty days and will not affect hospitals or private physicians.

Three Million Red Cross Volunteers to Continue War Work

Mrs. Mason Colt, new national administrator of the National Red Cross Volunteer Special Services, announces that about three fourths of the 4,000,000 wartime volunteers plan to continue Red Cross work as a permanent community service. Her estimate was given following a four day conference in the Hotel Statler with 150 delegates from thirty-nine states. She said there will be plenty of work for peacetime volunteers, as hospital nurses' aides, in health clinics, in blood banks, in tuberculosis, eye and tooth clinics and as producers of bandages and surgical dressings for local hospitals. Mrs. Colt denies that socialites dominate the Red Cross and that the organization had refused to cooperate with other welfare workers.

Inadequate Pay Keeps Doctors from the Veterans Administration

Gen. Omar N. Bradley, Veterans Administrator, reveals that the agency is 1,300 doctors short of its 2,300 goal, largely because of disparity in pay. He says that unless the service is made more attractive to doctors the situation will become worse, as 570 lent by the Army and Navy are to be discharged and returned to private practice. In addition to better pay, General Bradley advocates (1) giving veterans doctors' recognition and (2) giving them a chance to improve and practice the most modern medicine. These were improvements in practice for Veterans Administration doctors which General Hawley, acting Surgeon General, said recently would be made.

Medicine and Food Dropped in New Cargo Bomb

How medicine and food can be dropped to communities cut off by flood, forest fire and other disasters was demonstrated at Bolling Field by the Army Air Forces when six wooden, snub-nosed "cargo bombs" were dropped at heights ranging from 200 to 1,000 feet. Two were dropped in water to show their buoyancy. The contents included rations, oil, gasoline, blood plasma, serums and first aid kits, all undamaged by the impact. The demonstration was staged for the American Red Cross in cooperation with the United States Steel Corporation, which developed the container with the Air Technical Service Command.

Council on Medical Service and Public Relations

PREPAID MEDICAL CARE NEWS

Latest on Prepayment Plans

Virginia.—The Virginia Medical Service Association, approved by the Virginia State Medical Society, is governed by a board of thirty-five persons, of whom not less than two thirds must be medical men. All administrative procedures are handled by the Blue Cross (Virginia Hospital Service Association). It is a combination indemnity-service plan with income limits for the latter set at $2,000, $2,500 and $3,000. A single medical-surgical contract is issued. The medical coverage is limited to in-hospital cases of three days' or more duration and pays up to thirty-five days a year. A sum not to exceed $10 a year is paid toward diagnostic x-ray in surgical cases. The surgical-obstetric coverage is similar to that of other society sponsored plans.

Maryland.—A plan has been drawn up and passed by the Maryland legislature. The details are now in the hands of Dr. Victor F. Cullen, State Sanatorium, Md.

Oklahoma.—The Oklahoma Physicians Service, approved by the Oklahoma State Medical Association, is now in operation throughout the state. There are fifteen trustees, of whom nine are doctors. The plan offers a surgical and obstetric indemnity contract with no medical provisions or riders.

Alabama.—The Alabama State Medical Association has turned to the Hospital Service Association (Blue Cross) of Alabama for medical-surgical coverage. The state laws were amended to enable the Hospital Service Corporation to provide medical and surgical contracts for its subscribers. The contracts provide an indemnity only and include inpatient medical care ($2 a day for twenty-five days), the usual surgical procedures and a number of additional in-hospital services not found in other plan contracts.

Arizona.—The governor of Arizona signed a bill on October 3, duly passed by the state legislature, entitled "the Arizona nonprofit hospital and medical service corporation law." This enabling act differs from the enabling acts of most states in that it provides that the corporation may sell either hospital or medical service contracts or both. It also provides for service contracts only, without provisions for indemnity payments.

Washington.—The Washington State Medical Bureau has been instructed to prepare a statewide contract suitable to cover such organizations as the grange, small farmer groups and business concerns which operate over a wide territory. Consideration will be given to the necessity for a broader contract, for income limitations and to the eligibility requirements for such groups. The bureau was reorganized in April of this year to coordinate the various local Medical Service Corporations operating in this state. Mr. Ralph W. Neill is the bureau manager and Dr. John F. Christensen the president.

New York.—The house of delegates of the Medical Society of the State of New York adopted a resolution on October 9 urging the organization of a national casualty company to develop voluntary medical insurance coverage in areas where none exists and to enroll nationwide employers' groups.

Tentative plans call for a stock company owned and controlled by the medical profession. The company would assist medical society-sponsored plans through national enrolment of nationwide concerns. It would provide coverage in areas where it is not available and remain only until such time as the profession could set up its own plan. Hospital coverage would be offered through Blue Cross.

The Blue Cross hospital groups are already operating a plan along this same line. However, instead of a new corporation, a central enrolment office has been organized with headquarters in New York. This office will service all eighty-seven Blue Cross Plans and merely act as agent for them.

Genesee Valley Prepayment Plan

Rochester physicians have officially approved a prepayment medical care plan to start operations on Jan. 1, 1946. The announcement was made by Mr. George P. Farrell, director of the Bureau of Medical Care Insurance of the Medical Society of New York.

The plan, Genesee Valley Medical Expense Indemnity Inc., is fully organized and awaits only the approval of the State Department of Social Welfare. It has its own trustees and will offer a limited surgical and obstetric contract until sufficient experience is gained to allow increased benefits.

Administrative service such as enrolment, billing and record keeping will be provided by the Rochester Hospital Service Association. The charge will be based on cost. All policies relating to the medical care plan will be determined by the Genesee Valley Medical Expense Indemnity Inc.

Coming Medical Meetings

American Medical Association House of Delegates, Chicago, Dec. 3-6. Dr. Olin West, 535 N. Dearborn St., Chicago 10, Secretary.

American Academy of Allergy, Chicago, Dec. 10-11. Dr. Karl D. Figley, 316 Michigan St., Toledo 2, Ohio, Secretary.

American Association on Mental Deficiency, Cleveland, Nov. 28-Dec. 1. Dr. Neil A. Dayton, Box 51, Mansfield Depot, Conn., Secretary.

American Society of Anesthetists, New York, Dec. 12-13. Dr. McKinnie L. Phelps, 745 Fifth Ave., New York 22, Secretary.

International College of Surgeons, U. S. Chapter, Washington, D. C., Dec. 7-8. Dr. Louis J. Gariepy, 16401 Grand River Avenue, Detroit, Secretary.

North Pacific Pediatric Society, Portland, Ore., Dec. 1. Dr. Aldis B. Johnson, Cobb Bldg., Seattle 1, Washington, Secretary.

Oklahoma City Clinical Society, Oklahoma City, Nov. 26-29. Dr. Elmer R. Musick, 512 Medical Arts Bldg., Oklahoma City, Secretary.

Puerto Rico, Medical Association of, San Juan, Dec. 14-16. Dr. Rafael A. Vilar, P. O. Box 3866, Santurce, Secretary.

Southern Surgical Association, Hot Springs, Va., Dec. 4-6. Dr. Alfred Blalock, Johns Hopkins Hospital, Baltimore 5, Secretary.

Medical News

(PHYSICIANS WILL CONFER A FAVOR BY SENDING FOR THIS DEPARTMENT ITEMS OF NEWS OF MORE OR LESS GENERAL INTEREST; SUCH AS RELATE TO SOCIETY ACTIVITIES, NEW HOSPITALS, EDUCATION AND PUBLIC HEALTH.)

CALIFORNIA

Neuropathologist for Langley Porter Clinic.—Dr. Nathan Malamud, Ann Arbor, Mich., has been appointed neuropathologist at the Langley Porter Clinic and associate clinical professor of psychiatry at the University of California Medical School, San Francisco. According to the University of California *Clip Sheet*, while an excellent neuropathologic laboratory was constructed when the clinic was built, this is the first appointment of a neuropathologist, which, it is expected, will further the plans to attack the problem of mental disease from every angle. Neuropathologic material will be received from all the state hospitals, states the *Clip Sheet*. The Langley Porter Clinic will not only be coordinated with the medical school of the university but will be the focus for these studies for the state hospital system, thus enabling the clinic to enlarge its service to the state. Dr. Malamud graduated at McGill University Faculty of Medicine, Montreal, in 1930.

New Medical School Planned.—Plans for the establishment of a new medical school in Los Angeles were under way October 29 following action of the regents of the University of California authorizing Robert G. Sproul, president, Berkeley, to seek funds from the state legislature for the purpose. Recognizing the need for additional facilities in the southern part of the state, the regents unanimously approved a recommendation to provide full training in medicine in connection with the Los Angeles campus of the university. Only one third of the doctors needed annually in southern California can be trained by the existing medical school there, according to the University of California *Clip Sheet*. University officials estimated that some sixty students would receive instruction in the new school each year, which would nearly equal the present facilities of the university on the San Francisco campus. Depending on action by the legislature, it may be from two to three years before the full curriculum can be offered in medicine, but lower division work may begin within a year, officials said.

COLORADO

Plan Medical Research Center.—Establishment of a major medical research center in Denver as a cooperative venture of the University of Colorado School of Medicine, Denver, the University of Denver and other institutions is now being considered, according to a statement to the press by Robert L. Stearns, LL.D., president of the University of Colorado. One branch would be designed to further medical science and improve public health in the area and would be operated in connection with the Colorado medical school and the Colorado General Hospital.

INDIANA

John N. Hurty Service Award.—The Indiana State Board of Health has created the John N. Hurty service award to honor the late physician who was secretary of the board from 1896 to 1922. Presented for the first time this year, the award was given to about 150 state employees who have been connected with water supplies and who have served twenty-five years or more. In this particular award Dr. Hurty is being honored for his efforts to organize the Indiana section of the American Water Works Association, which he served as president for two consecutive years.

State Medical Election.—Dr. Floyd T. Romberger, Lafayette, was chosen president-elect of the Indiana State Medical Association during its annual meeting in French Lick, November 6-8. Dr. Jesse E. Ferrell, Fortville, was installed as president. Dr. George R. Dillinger, French Lick, resigned as delegate. Dr. Alfred S. Giordano, South Bend, was elected delegate to the 1945 session of the House of Delegates of the American Medical Association and Dr. Don F. Cameron, Fort Wayne, was reelected delegate. Other delegates include Drs. Franklin S. Crockett, Lafayette, and Homer G. Hamer, Indianapolis. The 1946 meeting of the state association will be held in Fort Wayne.

MASSACHUSETTS

Lectures on Epilepsy.—Dr. William G. Lennox, Boston, opened a series of six free lectures on "Epilepsy, the Hopeful Disorder," November 15. The lectures are being given on Thursdays at the Boston Public Library under the auspices of the Lowell Institute.

Civilian Blood Program for Massachusetts.—The Massachusetts Department of Public Health will soon begin distribution of blood plasma and other blood products without cost on the same basis as serums and vaccines. The service, which is to be available to the citizens of the state, will operate as a unit of the division of biologic laboratories. The legislature has appropriated $174,000 to equip, staff and operate a blood and blood derivatives program, and the Godfrey M. Hyams Trust of Boston has donated $176,000 to Harvard University to construct a modern well equipped laboratory building in which processing and fractionation of blood and its products can be carried out as well as other essential procedures. The project will include collection of blood donations as donor programs are organized by civic or other groups in various communities; processing of the blood to the various fractions of known value, such as plasma, albumin, gamma globulin, fibrin foam, fibrin film, blood grouping serums and other fractions still in the process of development, as well as resuspended red cells and whole blood as soon as suitable methods for their preservation are established, and distribution of these products through recognized channels and in accordance with community contribution of blood donations.

MICHIGAN

Foundation for Medical and Health Education.—On September 19 the Michigan Foundation for Medical and Health Education, sponsored by the Michigan State Medical Society, was incorporated. Purposes of the foundation are to "acquire, provide, use, develop, endow and finance methods, means and facilities for postgraduate education in medicine, for education in medicine, for lay health education and for research, fellowships and scholarships." Officers of the foundation include Dr. Earl I. Carr, Lansing, president; Dr. Burton R. Corbus, Grand Rapids, vice president, and Herbert H. Gardner, president of the Birmingham National Bank, treasurer. William J. Burns, LL.B., Lansing, executive secretary of the state medical society, is secretary. In an effort to expedite and reach the goal of $150,000 within a year, Dr. Arthur S. Brunk, Detroit, retiring president of the society, offered in September to contribute the sum of $1,000 to the foundation provided ninety-nine members of the society contribute a like sum during the next twelve month period. This proposed $100,000 would be added to the state society's contribution of $10,000 and that of the late Dr. and Mrs. Arthur P. Biddle, about $40,000, to make the total by September 1946 of $150,000, the income of which would be available for immediate postgraduate needs. With the $33,000 subscribed within a month of this offer, the foundation on October 23 had $83,000. The Biddle bequest in 1944 was added to the $10,000 fund of the state society, which in 1942 established a graduate education program to stimulate postgraduate work among practicing physicians (THE JOURNAL, Aug. 15, 1942, p. 1380; Oct. 28, 1944, p. 578). Credit goes to Dr. Corbus, Dr. Carr and Dr. James D. Bruce, Ann Arbor, for establishing the new foundation.

NEW YORK

Experimental Test of Ultraviolet Rays in Children's Diseases.—The village of Pleasantville was recently chosen for the first communitywide test in the nation of the use of ultraviolet rays to disinfect air inhaled by school children. Beginning January 1 and continuing for three years, ultraviolet lamps will be used in all classrooms of the high school and two grammar schools of Pleasantville. Tentative arrangements have been made to install similar lamps in the motion picture theater, the six churches and other buildings where children congregate. The project is sponsored by the Westchester County Health Department, of which Dr. William A. Holla is director, the Milbank Memorial Fund of New York, the University of Pennsylvania School of Medicine, Philadelphia, and the General Electric Company. According to the *Bulletin* of the Medical Society of the County of Erie and the Buffalo Academy of Medicine, the experiments will provide radiant disinfection of air, with lamps concealed so as not to cast rays directly on the children. The *Bulletin* states that for the last nine years similar experiments have been conducted

in certain schools of Pennsylvania and adjacent states. The experiments are intended to destroy germs that cause measles, mumps, chickenpox, virus pneumonia and common colds, but little effect was anticipated in checking infantile paralysis.

Urge Reconstitution of State Medical Commission.—The house of delegates of the Medical Society of the State of New York, at its recent meeting, adopted a resolution requesting the governor "to reconstitute the New York State Commission on Medical Care by the appointment of additional active practitioners of medicine." It was also voted to request the governor's commission to "provide for a continuing conference with representatives of the society" in order that a plan may eventually be developed which will accomplish the declared objectives of the governor's charge to his own commission. The state society believes that "the tentative plans developed by this commission, as revealed recently to representatives of the Medical Society of the State of New York, contain many proposals and features which, in their judgment, are unpractical and would prove detrimental to sound medical progress and would effectually destroy the integrity and freedom of the medical profession." This last phrase has reference to the governor's charge to this commission, namely that it devise "a workable plan for broadening the availability of medical services and hospitals while at the same time preserving the integrity and freedom of the medical profession." The society also contends that this commission, as at present constituted, "contains an inadequate number of physicians who truly represent the general practice of medicine."

New York City

Spanish Speaking Association Plans Congress.—The International and Spanish Speaking Association of Physicians, Dentists and Pharmacists is making plans for a postwar Latin American congress, according to the secretary, Dr. Ernest F. Dupre. Dr. Jacob M. Gershberg, president, recently returned from a trip to Mexico, Central America and San Francisco during which he added seven new chapters, bringing the total of such units in the association to forty-nine.

Meeting of Society to Study Blood.—The recently formed Society for the Study of Blood met at the New York Academy of Medicine November 8 to hear the following speakers: Drs. John T. Edsall, Boston, on "The Antihemophiliac Globulin of Normal Plasma," Louis K. Diamond, Boston, "The Clinical Use of an Antihemophiliac Fraction of Blood Plasma," and Randolph West, New York, and Erwin Chargaff, Ph.D., New York, "Hemophiliac Clotting Defect in a Female." The society was formed at a meeting June 14 (THE JOURNAL, July 21, p. 893).

Clinical Aspects of Visceral Insufficiency.—Members of the staff of Mount Sinai Hospital are offering a series of lectures on "The Clinical Aspect of Visceral Insufficiency." The series follows:

Dr. Eli Moschcowitz, November 28, Intestinal Insufficiency. The Sprue Syndrome.
Dr. Ernst P. Boas, December 12, Myocardial Insufficiency.
Dr. Charles K. Friedberg, December 26, Peripheral Circulatory Insufficiency.
Dr. Arthur M. Fishberg, January 9, Renal Insufficiency.
Dr. Benjamin Eliasoph, January 23, Thyroid Insufficiency.
Dr. Coleman B. Rabin, February 6, Respiratory Insufficiency.
Dr. Isidore Snapper, February 20, Parathyroid Insufficiency and Osseous Disease.
Dr. Louis J. Soffer, March 6, Adrenal Insufficiency.
Dr. Henry Dolger, March 20, Pancreatic Insufficiency.
Dr. Solomon S. Lichtman, April 3, Hepatic Insufficiency: Metabolic Aspects.
Dr. Joseph H. Globus, April 17, Pituitary Insufficiency.

OHIO

Training for Returned Service Physicians.—Western Reserve University School of Medicine, the Academy of Medicine of Cleveland and eighteen hospitals, comprising the Cleveland Hospital Council, will cooperate in providing special training for three different groups of doctors in need of refresher courses. The first phase of the program will be handled by individual hospitals, while additional internships and residencies will be provided within the capacities of the clinical facilities at each. The second phase of the overall program has been developed for ex-army and navy physicians who have completed two or more years of hospital training, with the medical school offering a three month refresher course covering fields of medicine, pediatrics and general surgery. Students may enroll in the refresher course on the first of any month in which there is a vacancy. The program will include a series of seminars in various topics, with free time allowed for reading and consultation with members of the staff. The third phase of the program is a "graduate fortnight" for both former military medical officers and the civilian doctor who has been too hard pressed during the war years to keep up with latest developments. This is under a special committee of the academy of medicine.

OREGON

Changes in Health Officers.—Dr. Robert W. Ripley has resigned as health officer of Corvallis to accept a position in the Connecticut State Department of Health. Dr. William T. Edmundson, Newberg, has been appointed health officer for Hood River County, effective August 1, to succeed Dr. Cecil W. McCain, Hood River, resigned.

New Director of Industrial Hygiene.—Dr. Charles M. McGill, New Orleans, surgeon, U. S. Public Health Service, has been appointed director of the industrial hygiene division of the Oregon State board of health, effective October 1. Dr. Thomas F. Mancuso, Portland, who has been acting director, has been assigned to a similar position with the Ohio State Health Department, according to the Portland *Journal*, September 12.

PENNSYLVANIA

Graduate Assembly.—The Harrisburg Academy of Medicine will sponsor a postgraduate assembly November 29. The following members of the staff of Harvard Medical School, Boston, will participate: Dr. Charles A. Janeway, assistant professor of pediatrics, Dr. Lewis Dexter, instructor in medicine, and Dr. Sinclair Armstrong, instructor in medicine and research associate in physical chemistry.

Philadelphia

Schireson Injunction Against State Board Dissolved.—The injunction restraining the Pennsylvania State Board of Medical Education and Licensure from revoking the license of Henry Junius Schireson, allegedly obtained by fraud, was dissolved by Judge Karl E. Richards in the court of common pleas of Dauphin County, November 2. Through this action the court upheld the contention of attorneys for the board that a state licensing body had "the inherent right to revoke a license obtained by fraud." The court also refused to accept the plaintiff's contention that the board was guilty of laches in attempting to revoke a license which had been issued some thirty years earlier. In explaining the latter decision the court pointed out that the mere passage of time is not a criterion in determining the guilt of laches. Since the record did not disclose when the board became aware of the fraudulent aspect of the license, the court could not find any basis for holding the board guilty of laches. On June 29, 1910 Schireson was issued a license authorizing him to practice medicine and surgery in the state. Under this license he practiced in Pennsylvania from 1910 to 1913 and again from 1932 to the present time. On June 30, 1944 the state board of medical education and licensure issued a citation to Schireson requiring him to show cause why his license should not be revoked for alleged fraud, misrepresentation and deception practiced by him in procuring his license. Before the time set for hearing on the citation, Schireson filed a bill of complaint seeking to restrain the board from proceeding thereon. A preliminary injunction was granted, which by consent of the parties had been continued to the recent hearing.

VIRGINIA

Personal.—Dr. James N. Dudley, for some time health officer for the Southside Health District with headquarters at Farmville, has been appointed health officer for the city of Newport News; he was to have assumed office on October 1. He succeeds Dr. William Y. Garrett, Eastville, who resigned because of his health.——Dr. Thomas D. Walker Jr., Newport News, has been appointed city physician of Newport News to succeed Dr. Louis Loeb, retired.——Dr. Albert T. Brickhouse, health officer of Hopewell, has been appointed city coroner to fill the unexpired term of Dr. Johannes C. Bodow, resigned, it is reported. Dr. Brickhouse will serve until Dec. 31, 1947.

State Medical Election.—Dr. William L. Powell, Roanoke, was made president-elect of the Medical Society of Virginia at its meeting in Roanoke October 23. Dr. Julian L. Rawls, Norfolk, was installed as president. Other officers include Drs. James D. Hagood, Clover, William L. Peple, Richmond, and Walter C. Caudill, Pearisburg, vice presidents. Miss Agnes V. Edwards, Richmond, was reelected secretary. The society voted to hold its 1946 meeting at Virginia Beach in October. Dr. Henry B. Mulholland, Charlottesville, retiring president of the society, was named delegate to the American Medical Association.

WASHINGTON

Bubonic Plague in Tacoma.—The city of Tacoma is said to be again "free of plague infection" through a concerted program of rodent control. During the past three years the port of Tacoma has been in quarantine about twenty-one months and in this period has been quarantined and reopened twice (THE JOURNAL, January 6, p. 46). According to *Northwest Medicine* the statement "free of plague infection" came from the U. S. Public Health Service, and control of the disease is again in "the hands of Tacoma."

Personal.—Dr. Ann P. Kent, Washington, D. C., has joined the staff of the Territorial Department of Health as field physician in charge aboard the mobile health unit vessel *Hygiene*.——Dr. Edward L. Van Aelstyn, Price, Utah, has been appointed director of the Cowlitz and Wahkiakum counties health departments, succeeding Dr. Thomas H. Biggs, Kelso, who resigned recently to devote his time to private practice.——Dr. Walter M. Morgan has been named health officer of Kent, succeeding Dr. George M. MacGregor, who resigned June 1.

Emily Pratt Named Head of New Hearing Section.—Dr. Emily A. Pratt, Albany, N. Y., who was appointed as otologist with the Washington State Department of Health, October 1, will be the head of the newly created Conservation of Hearing Section. Dr. Pratt, who has had years of successful experience in developing services for hard of hearing children in New York, will intensify the program now being conducted for school children in the state of Washington. This work will be carried out in cooperation with the department of public instruction and will fulfil the provisions of the law passed at the last session of the state legislature.

WEST VIRGINIA

Personal.—The following physicians were recently elected to honorary life membership in the West Virginia State Medical Association: Drs. Charles M. Hawes, Huntington; Raymond M. Sloan, Huntington; Henry E. Baum, Charleston; Wilbert G. Drinkwater, Gormania, and Jesse O. Bailiff, Mullens.

Cancer Field Worker Appointed.—Mrs. Frances Meyer, Charleston, has been appointed cancer field worker for Kanawha County under the sponsorship of the local chapter of the Field Army of the American Cancer Society. The project, which is the first of its kind to be undertaken by the Field Army in West Virginia, will be paid for from funds raised during the 1945 cancer drive. Mrs. Meyer, who is experienced in the field of public welfare, will do follow-up work with needy cancer patients to make sure that they return to their doctors for follow-up examinations. She will cooperate with and aid city and county health nurses, the health department and the local department of public assistance to utilize all services available for obtaining care for needy cancer patients.

State Association Plans New Home.—Initial steps looking toward the construction of a permanent home for the West Virginia State Medical Association were taken by the council at a meeting at Parkersburg October 14. The council directed the president, Dr. Thomas L. Harris, Parkersburg, to appoint a committee to confer on the subject with a similar committee from the Kanawha Medical Society. Recognizing the need for the extension of medical care to outlying districts in the state, the council directed the president to appoint a committee on rural medical service to work with committees of the American Farm Bureau Federation and other farm organizations planning rural health improvement. Besides approving revisions by the Public Health Council of regulations concerning the control of communicable diseases, the council recommended the adoption in principle by that body, as the official communicable disease code for West Virginia, of the American Public Health Association's manual on "The Control of Communicable Diseases."

WISCONSIN

Surgical Meeting.—The Wisconsin Academy of Surgery held a joint meeting with the Jackson Clinic Post-Graduate Group at Madison October 18-20. Among the speakers were:

Dr. George H. Ewell, Madison, Polycystic Kidney.
Drs. Roger E. Henning, Madison, and Arnold S. Jackson, Madison, The Management of Gallbladder Disease.
Drs. Harold E. Marsh, Madison, and James A. Jackson, Madison, The Problem of Ulcer.
Dr. Arthur C. Stirling, Madison, Ruptured Intervertebral Disk.
Dr. Donald L. Dickerson, Danville, Ill., Penicillin and Streptomycin in Surgery.
Dr. Luther E. Holmgren, Madison, The Hernia Problem.

A special lecture was given by Dr. Warren H. Cole, Chicago, on "Treatment of Surgical Lesions of the Stomach Including Preoperative and Postoperative Care."

GENERAL

New Journal on Geriatrics.—The new bimonthly medical journal *Geriatrics*, devoted to research and clinical reports on the processes and the diseases of the aged and aging, will appear in January, Modern Medicine Publications announced. The editor will be Dr. Axel E. Hedback, Minneapolis.

Luzier Grant for Work in Dermatologic Allergy.—The American College of Allergists announces a recent grant of $1,500 to the college to be used for a research fellowship from Luzier's, Inc., Kansas City, Mo. The grant is to be known as the Luzier Grant for Fundamental Investigation in the Field of Dermatologic Allergy. The fellowship will be under the direction of Drs. Rudolf L. Baer, New York, and Stephan Epstein, Marshfield, Wis.

Cancer Society Opens Midwest Branch.—Mr. George B. Larson, formerly assistant secretary of the Wisconsin State Medical Society and for the past year associated with the Field Army of the American Cancer Society, has been named Midwest Field Army director in charge of a new midwest branch to be opened in Suite 1512 of the Marshall Field and Company Annex Building, 25 East Washington Street, Chicago. The new office will supervise the states of Illinois, Indiana, Iowa, Michigan, Minnesota, Missouri, Wisconsin, Kansas and Arkansas.

Allergy Meeting.—The American Academy of Allergy will hold its second annual meeting at the Palmer House, Chicago, December 10-11, under the presidency of Dr. Oscar Swineford Jr., Charlottesville, Va., who will discuss undergraduate education in allergy. Among the speakers will be:

Drs. Harold D. Dundy, Benjamin Zohn and Robert Chobot, all of New York, Clinical Evaluation of Histamine Azoprotein.
Dr. Albert V. Stoesser, Minneapolis, Influence of Soy Bean Products on the Plasma Lipids and the Course of Eczema.
Dr. Jehu M. Robison, Houston, Texas, Physiology and Functional Pathology of the Lymphatic System Applied to Allergy of the Nose and Paranasal Sinuses.
Dr. Frieda Baumann, Dr. Jean Crump, Dr. Lloyd D. Seager, Dr. Ann Catherine Arthurs and Ruth E. Miller, Ph.D., all of Philadelphia, Use of Oral Penicillin in Treatment of Intrinsic Asthma.

The program will also include a symposium on infectious asthma and one on antihistaminic and antianaphylactic drugs.

Advisory Health Group Meeting.—Prompt action in the development of an international health organization was urged by thirty leaders in public health and civic activities at a meeting held in the Department of State, Washington, D. C., October 11-12 on an advisory health group called by the department under the chairmanship of Dr. Thomas Parran, Surgeon General, U. S. Public Health Service. The group recommended that the U. S. government associate itself immediately with other nations in taking steps leading to the early formation of a new and broad international health organization to be closely linked with the Economic and Social Council of the United Nations. Certain general functions visualized for the organization are the collection and analysis of worldwide disease statistics as a basis for epidemic control, assistance to national health services to control disease at their sources, centralization, consolidation and subsequent distribution of health and medical knowledge and the standardization and control of drugs and other therapeutic agents.

Five Years of Rehabilitating Asthmatic Children.—The National Home for Jewish Children at Denver has for the past five years been caring for underprivileged children suffering from acute bronchial asthma and other refractory upper respiratory diseases. The home is located in Denver and is available to children from all over the country whose families cannot afford extensive and costly private care. The children remain until physicians recommend that they be returned to their homes. In a comparatively normal environment, and sharing the companionship of other children, the asthmatic child is under expert medical supervision at the home and is cared for by a staff trained in child welfare. During his stay, periodic examinations are made and full reports given to the referring physicians and hospital clinics. The National Home has a modern, well equipped infirmary where children requiring bed care are attended by registered nurses under the supervision of the attending physicians. The children are housed in attractively furnished congregate-cottages with individual bedrooms. Food of the highest quality is carefully prepared in modern kitchens, and special diets are given when prescribed by physicians. The children participate in the community life by attending the local schools and taking part in communal recreations. In addition to the intensive preparation made prior to the child's admission to the home, an equally thorough after-care program

is carried out on the return of the child to his family. The aim of the program is to make certain that the child will continue to enjoy his restored health in the very environment which originally was a factor contributing to the child's illness. Trained social workers educate the parents to the general health practices which play an important part in the treatment of allergic diseases. Application may be made by communicating with the Social Service Department of the National Home for Jewish Children at 1457 Broadway, New York 18, or through the office of the Jewish Social Service organization in any community.

Southern Surgical Meeting. — The fifty-seventh annual meeting of the Southern Surgical Association will be held at The Homestead, Hot Springs, Va., December 4-6 under the presidency of Dr. Charles A. Vance, Lexington, Ky., whose subject will be "The Transylvania Medical Library." Among the speakers on the program will be:

Dr. William P. Longmire Jr., Sapulpa, Okla., and Major Marcus M. Ravitch, M. C., Baltimore, A New Method for Constructing an Artificial Esophagus.
Major Robert P. Kelly, M. C., Capt. Louis M. Rosati, M. C., and Capt. Robert A. Murray, M. C., White Sulphur Springs, W. Va., Treatment of Traumatic Osteomyelitis.
Col. Daniel C. Elkin, M. C., White Sulphur Springs, Cirsoid Aneurysm of the Scalp.
Major Boris P. Petroff, M. C., Martinsburg, W. Va., Streptomycin in Treatment of Urinary Infections.
Drs. Charles S. Venable and Walter G. Stuck, San Antonio, Texas, Muscle Implant in the Treatment of Monarticular Hypertrophic Arthritis of the Hip (Destructive Arthritis, Aseptic Necrosis and Other Conditions).
Dr. J. Albert Key, St. Louis, Bunnell's Pull Out Wire Sutures for the Fixation of Tendons, Both as Grafts and as Transplants.
Lieut. Col. Brian B. Blades, M. C., Washington, D. C., Mediastinal Tumors: A Report of Cases Treated at Army Thoracic Surgery Centers in the United States.
Dr. Guy L. Hunner, Baltimore, Is Ureteral Stricture an Etiologic Factor in the Genesis of Renal Tumor?

One feature will be a symposium on "Newer Concepts in the Treatment of the Paralyzed Patient Due to Wartime Injuries of the Spinal Cord."

Fellowships in the Medical Sciences.—Fellowships in the Medical Sciences, similar to those which have been administered by the Medical Fellowship Board of the National Research Council since 1922, will again be available for the year beginning July 1, 1946. These fellowships, supported by grants from the Rockefeller Foundation to the National Research Council, are designed to provide opportunities for training and experience in research in all branches of medical science. They are open to citizens of the United States or Canada who possess an M.D. or a Ph.D. degree and are intended for recent graduates who are not yet professionally established. In addition to these fellowships the medical fellowship board administers two groups of research fellowships, made available through a grant from the National Foundation for Infantile Paralysis, Inc. The first group, open to applicants who hold either the Ph.D. or the M.D. degree, is for the purpose of providing opportunities for special training and experience in the study of filtrable viruses. The second group, open only to graduates in medicine who have completed one or more years of hospital experience in clinical surgery and are planning a career in orthopedic surgery, is designed to provide opportunities for training and research in those basic medical sciences which will be of particular value in furthering progress in the field of orthopedic surgery. A series of fellowships in anesthesiology has been established through a grant from the American Society of Anesthesiologists. These fellowships are offered with a view to fostering a closer union between the clinical practice of anesthesiology and the fundamental disciplines on which anesthesia rests. Applicants must hold the M.D. degree and must have completed one or more years of hospital experience as intern or resident. Fellows will be appointed at a meeting of the Medical Fellowship Board late in February 1946. Applications to receive consideration at this meeting must be filed on or before January 1. Appointments may begin on any date determined by the board. For further particulars concerning these fellowships, address the Secretary of the Medical Fellowship Board, National Research Council, 2101 Constitution Avenue, Washington 25, D. C.

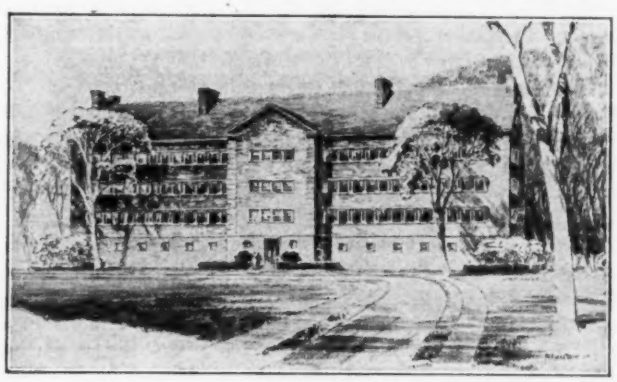
CORRECTION

Incorrect Affiliation.—Mr. Irvin R. Vaughn is director of the division of public health education and assistant director of the division of vital statistics of the South Dakota State Board of Health. THE JOURNAL, September 29, page 367, in its Organization Section, referred to Mr. Vaughn as being affiliated with the North Dakota State Health Department.

Government Services

Memorial Laboratory of Infectious Disease

Construction has started on a new infectious disease laboratory of the National Institute of Health, U. S. Public Health Service, and the Federal Security Agency. The building is located in Bethesda, Md., and will be known as the Memorial Laboratory of Infectious Diseases of the United States Public Health Service, in tribute to the laboratory workers of the service who died as a result of risks involved in their professional duties. New methods of control are to be employed. Forced ventilation, with pressure control of air intake and output, will be a major factor of protection. The correct placement of air vents and the control of air volumes and velocities are intended to insure a safe air supply. Air currents so controlled will provide escape for infectious matter in the air. Moreover, contaminated air will be purified by being drawn by fans through protective fluelike hoods. The hoods will contain electric grills that will automatically incinerate infectious particles. Automatic mechanisms which will not operate until sealed are additional safety innovations that will be used on high speed equipment. These machines will also be flooded with ultraviolet light. Workers will leave and enter the laboratories through "decontamination chambers." These chambers will be divided into three sections, a dressing room for street clothes, a shower room and another dressing room for laboratory clothes. Special mirror arrangements will be installed so that workers can thoroughly inspect themselves. The entire building will be sprayed with DDT, and



Infectious Disease Laboratory.

there are to be six separate self-contained units in two wings, which will be separated by a main hall, on which the clerical offices and library will open. These units will be dedicated to the individual study of infectious diseases. Each unit will contain a small laboratory room for chemical and serologic work and a larger room containing three small glass enclosed cubicles for the cutivation of the infectious agent. Unit laboratories will have special equipment rooms. In addition, the units will contain three animal rooms—one for large animals and the remaining two to house the small laboratory animals. Plans also call for a necropsy room, a hot and cold temperature room and a sterilization chamber. The units are to be wholly self contained and completely independent. Each will have its own dumbwaiter, which will connect directly with the incinerator. With an overall floor space of 9,626 feet, the three story building will be approximately 185 feet long and 48 feet wide. The interior is to be tile surfaced and of "flush" construction. All pipes, shelves, cabinets and lights are to be built into the walls and ceilings to facilitate cleaning and sanitation. The exterior will be finished in red brick to conform with the other six buildings located on the 97 acres of land of the U. S. Public Health Service. The original sum needed for the construction of the building was obtained by the public health service from the Public Building Administration with the approval of the Bureau of the Budget. While some of the equipment will be purchased out of annual funds of the National Institute of Health, it may be necessary, in order to equip the building completely, to secure additional funds. Present plans call for the completion of the building around May 1946. It is planned to open it for public inspection for two weeks, after which time the only visitors permitted entry will be scientists.

Foreign Letters

LONDON

(From Our Regular Correspondent)

Oct. 20, 1945.

The Disabled Persons Register

The measures taken by the government for the benefit of the disabled are on a scale never before attempted and are most thorough. An important new feature is the register of disabled persons entitled to the advantages provided by the Disabled Persons Employment Act. Persons disabled through war service, through industrial, road or other accident or congenitally disabled are entitled to register. Disablement due to disease is recognized equally with disablement caused by wounds or injury. Thus individuals who do not qualify for war disability pensions come within the scope of the act.

There are three principal benefits: (1) courses of vocational training and industrial rehabilitation for those who need to learn a new profession, trade or occupation or who require a special course to return to work after hospital treatment for injury or sickness, (2) assistance toward ordinary employment through requiring employers to engage an indicated proportion of disabled persons and (3) employment under special or sheltered conditions for the more severely disabled, who could not obtain work in competitive employment.

Registration is voluntary and its one purpose is to obtain a list of identifiable disabled persons. When this attains any size the minister of labor will fix the quota of disabled persons whom every employer of more than thirty persons must engage. To begin with, the quota will probably be 2 per cent, and as the register grows so also will the quota. It is expected that the register will grow to a million.

German Losses in the War 7,400,000

In the House of Commons the prime minister, Mr. Atlee, stated that the estimated total losses of the German armed forces in killed, permanently wounded and permanent medical casualties were 7,400,000. This is in striking contrast to the total losses of the British commonwealth and empire forces, which amounted to 1,233,796, of whom 336,772 were killed. This total includes 330,523 prisoners of war and covers the period from Sept. 3, 1939 to May 31, 1945.

In the war of 1914-1918 the British losses were much greater. The British total losses amounted to 3,286,090, of whom 966,230 were killed. The German losses were 6,252,494, of whom 2,050,466 were killed. The greatly reduced British casualties in the second and greater war may be explained by the greater amount of fighting on the Russian front than in the previous war and the excellent arrangements for the crushing of Germany on the western front. The artificial harbor for the landing on the Normandy coast, which was prepared in secrecy, came as a complete surprise to the German high command, who, with characteristic thoroughness, did everything possible to render impregnable the places where they thought a landing might be attempted. This device alone probably saved hundreds of thousands of casualties.

Total Prohibition of Opium in the Far East by the British Government

The total prohibition of opium in Malaya and all British protected countries in the Far East is announced by the British military administration in Singapore. This takes the place of the policy of Malaya before the war of gradually reducing government revenue from opium with total prohibition as the goal. The success of the new policy will depend on effective control of opium production in other countries. The British government is therefore consulting with other governments to secure cooperation.

SWITZERLAND

(From Our Regular Correspondent)

Oct. 26, 1945.

Franco-Swiss Medical Week

A Franco-Swiss Medical Week, organized under the auspices of the Swiss Academy for Medical Sciences, has just ended. It held its meetings in the University of Geneva. With profound emotion the Swiss medical group received 120 doctors from France. This was the first official contact between medical people of France and Switzerland since the armistice of 1940. Among the French guests were Professor Roussy, rector of the University of Paris, Professor Courrier of the Institut de France, Professor Brouardel, president of the French Medical Academy, Professor Broq, secretary of the French Surgical Academy, Professor Cornil, Professor Mondor, Professor Baudoin, dean of the Medical College of the Paris University, Professor Hermann, dean of the Medical College of the University of Lyons, and Dr. Cavaillon, general secretary of the Ministry of Public Health.

Professor Wegelin, president of the Swiss Academy of Medical Sciences, received the French guests at the official opening, where Messrs. Etter, minister for home affairs, Lachenal, Hoppenot, French ambassador in Switzerland, Professor Babel, rector of the University of Geneva, and Professor Bickel, dean of the Medical College of the same university were also present. Professor Roussy, rector of the University of Paris, and of Swiss origin, received the title of Doctor honoris causa of the University of Lausanne, together with Professor Charles Lenormand.

Tropical Institute in Basle

Thanks in part to facilities provided by the American authorities, a scientific expedition has left the Swiss Institute for Tropical Sciences in Basle for Leopoldville in the Belgian Congo, West Africa. This expedition aims to prepare for the hospitalization in Switzerland of patients suffering from tropical diseases. The mission, which includes Professor R. Geigy, director of the institute, Professor F. Rouet of the Pathologic Institute in Basle and Dr. H. Gashen of the Institute for Hygiene in Lausanne, will collect medical and entomologic data on tropical diseases, the causes and the agents through which they are transmitted and other data which will serve for research work and lectures at the Tropical Institute. The mission will remain some four months and then return to Switzerland.

Professor Mazel in Switzerland

Professor Mazel, director of the Institute of Forensic Medicine at the Medical College of Lyons, spoke at Geneva and Lausanne concerning the German occupation in the Lyons area and its medical consequences. Professor Mazel, a personal victim of the Nazis, as he was imprisoned in the Fort de Montluc, described with great dignity and perfect scientific objectivity the sufferings of Lyons and France. The practice of medicine during the occupation was difficult not only because of the lack of materials but because the occupation itself was trying particularly when health certificates had to be delivered for the exiled workers in Germany. It became even more difficult when the doctors were ordered to give to the Pétain authorities the names of people wounded by bullets; it was not in accord with professional ethics and it meant that people in the resistance movement would be arrested. Fortunately the National Council of the Order of Doctors intervened and that order was revoked.

Professor Mazel said that the only good things resulting from the occupation was the decrease of alcoholism and crime. This was due to the difficulty of obtaining alcoholic drinks and to the fact that most of the young people had gone to Germany or to the Maquis. On the other hand, criminal acts by boys

and girls, such as thefts and delicts due to disorganization of the family increased. The same applies to political criminality. Professor Mazel had been officially appointed to make an inquiry into the crimes committed by the Nazis in the Lyons area.

American Doctors in Switzerland

An officer from the headquarters of General Eisenhower is considering with Swiss authorities the possibility of students in uniform studying in Switzerland. About 150 doctors and nurses would come for one or two weeks to visit Swiss hospitals and sanatoriums. Lectures during one to two or three months would also be organized in the different medical colleges for 2,500 students.

GERMANY

The following medical news has been obtained through the Department of Commerce, Washington, D. C.—EDITOR.

Work Being Done at the University of Munich Medical School

The pharmacology department had an enrolment of 400 to 600 students per semester in normal times. The last instruction period ended about March 1, 1945, but previous damage to the buildings and equipment prevented practical work from being carried out during the winter semester. Prof. Walther Straub, former head of the department and the leading pharmacologist in Munich, died in October 1944 at the age of 70. He was succeeded by his chief assistant, Dr. A. W. Forst. Professor Straub had been interested in the use of copper sulfate for phosphorus burns and narcotine as a substitute for codeine. It was stated that all this work had been published. In addition to teaching, one of the chief functions of the department was the standardization of digitalis and strophanthin preparations. This work was originally shared by Berlin, Leipzig and Munich as the result of a government order issued in 1928. Recently, owing to the overwork of Berlin and the bombing of Leipzig, this function fell entirely to Munich. Since the occupation, Munich has also ceased to perform this duty. The methods used were the frog method (animals kept at a constant temperature) and the Knafl-Lenz method of infusion into guinea pigs, with constant observation of the electrocardiographic changes. The firms for which standardization was performed included Merck, Ysatfabrik in Wernigerode and Hageda in Berlin. A small amount of private work had also been done for the Luitpoldwerk of Munich on the toxicity of styptics, one of which was an alcohol-ether extract of lung called Clauden (also used clinically by Prof. Dr. Wilhelm Stepp). Dr. Forst's private scientific interests revolved principally around the autonomic nervous system in relation to sleep, following largely on the ideas of Hess of Zurich. Dr. Forst stated that no work of military significance had been undertaken and that no new classes of synthetics had been studied. The department has been largely wrecked, all that is left being the lecture theater (slightly damaged) and a practical chemical laboratory at present without water or gas. The equipment for the testing of digitalis and strophanthin had been evacuated to Tutzing, about 25 miles southwest of Munich.

MEDICAL CLINIC

Prof. Dr. Wilhelm Stepp, the director of the medical clinic, stated that his interests have been mainly in the field of vitamin therapy. In addition to his discussion of the vitamin B complex and his advocacy of its use as a whole, he mentioned particularly the successful use of pantothenic acid in doses of 50 to 100 mg. daily by injection or by mouth in a limited number of cases of chronic bronchitis. He also suggested the study of its use in pneumonia. His supplies of pantothenic acid came from Hoffmann-LaRoche at Grenzach. He also mentioned the use of nicotinamide in chronic gastroenteritis and colitis. In 1944 Stepp lectured in Spain and Portugal on rational vitamin therapy. He mentioned that the incidence of polyneuritis had greatly increased in the Munich area, particularly since 1939, especially among adults. Diphtheritic polyneuritis had also increased.

Stepp expressed an active interest in the employment of citrin in hemorrhagic diathesis and hemorrhagic retinitis and mentioned favorable clinical results. He had no knowledge of any methods for the standardization of citrin. In these conditions he often used vitamins C and K in conjunction with citrin. The citrin was supplied by I. G. Farbenindustrie. The gold compound Solganal is still being used in arthritis. He maintained that the blood substitute "Periston" was giving good results and stated that no liver damage had been observed. A few persons out of a group of 18 patients who had recently been sent to the clinic from Dachau were receiving Periston. He volunteered the information that he "had had no idea of the condition of affairs at the Dachau camp but had been under the impression that prisoners were employed on cultivation of medical plants, under good conditions of sunlight and air." Stepp referred to a stringent shortage of the purine alkaloids in Germany. He also mentioned the new (sixth) edition of Die Vitamine by Stepp, Kühnau and Schroeder, published in 1944 by Enke at Stuttgart. Stepp stated that Prof. Hans Fischer had recently committed suicide.

Anatomy Department—Division of Experimental Biology. Prof. Benno Romeis was not seen, as he had been evacuated from Munich to Diessen on the Ammersee. A copy of his book Blutgefäss- und Lymphgefässapparat innersekretorischer Drüsen, part 3, Innersekretorische Drüse II Hypophyse (Berlin, Julius Springer, 1940) was examined. It was well documented and illustrated but contained no matters of interest with regard to glandular preparations. Professor Bauer, who was the only staff member present, stated that the normal complement of students in the school of anatomy was approximately 600.

DERMATOLOGY DEPARTMENT

The head of the dermatology department, Dr. Julius Mayr, was absent at Starnberger See. His deputy, Dr. Hans Otto Münsterer, stated that he had been interested in vaccinal immunity in rabbits, but this work stopped during 1941 because of a lack of food for the animals. He reported that there had been relatively little occupational disease in the area, the main ones reported being cases of dermatitis due to light metals and hydrocarbon oil. Among outpatients a great increase in the incidence of pyoderma had been encountered. This condition was treated mainly with sulfonamides, particularly sulfathiazole. The sulfonamides have been in short supply and practically unobtainable recently. There has also been a scarcity of the arsphenamines, but the supply of bismuth preparations was adequate. In the treatment of syphilis practically no cases of jaundice were encountered, and on inquiry it was found that the syringes were boiled for each case. The incidence of syphilis in the war years was said to have increased more than tenfold. Dr. Münsterer stated that he had found a considerable incidence of venereal disease among foreigners in the area, except among the Russians. Scabies had increased. The preferred treatment was with "Mitigal." Benzyl benzoate was irritant and less effective. It does not appear that the civilian population has benefited directly from any advances in military medicine. It seems that drugs in short supply were reserved for the German army.

PSYCHIATRIC CLINIC

Prof. Oswald Bumke was visited in the Psychiatry Clinic in the Nürnberg Strasse. He gave a rapid summary of the work in his department. He himself had done little or no scientific research during the war. During this period, however, he had brought out the sixth edition of his Lehrbuch der Geisteskrankheiten, which is basically the same as the fifth edition. In the introduction to the fifth edition it was stated that the insulin-

metrazol-electric shock treatment would be introduced. In accordance with the new law the chapter on the state and the mentally ill was radically changed. Its relations to psychoanalysis were omitted. This was written in August 1941. Professor Bumke has also written a psychologic work, Gedanken über die Seele (Reflections on the Psyche) Julius Springer, of which there have been several printings, but a copy of this was unobtainable. He said it was psychologic and philosophical in content. Since the autumn of 1940 he had not been able to do any research because of his numerous clinical duties. Of his assistants and colleagues he mentioned one, Bannwarth, who had to go through the formality of joining the S. S. in order to become a dozent. Bannwarth had written a monograph entitled Ueber entzündliche Erkrankungen des Nervensystems (Inflammatory Diseases of the Nervous System). Among the subjects treated was inoculation polyneuritis. Because of the description of nervous symptoms following some of these inoculations, publication was held up by the censor so that it might not be used for enemy propaganda. Bumke's comments were acid. This worker apparently specialized in roentgenography of the general nervous system, and encephalography. The apparatus which he used has been evacuated to Tegernsee. One of Bumke's former colleagues in the Kaiser Wilhelm Institut, Professor Beck, has completed a monograph on the cytoarchitectonics of the brain. This was published about 1941 by Springer. Professor Beck is now at Tutzing working in an observation field hospital. His interests seem mainly anatomic. Another of Bumke's assistants, Dr. Scheid, had been working on metabolism in insanity. Unfortunately this worker was shot by the S. S. about the time the Americans arrived in Tegernsee, to which he had been evacuated. Scheid had been implicated in the Bavarian freedom movement. Professor Bumke was of the opinion that biochemical studies of metabolism in insanity would be much more profitable than anatomic studies. An article, or perhaps a book, on Psychogenische Störungen bei Kriegsteilnehmer (Psychogenic Disturbances Among Servicemen) had been written by D. Ziehen, another of Professor Bumke's group. A copy of the manuscript had been sent to Berlin, but publication was forbidden, and Professor Bumke did not know whether there was another copy. However, Ziehen, who is at present at a hospital in Haar, may have an original typescript.

No work had been done with the electroencephalograph in Bumke's institute during the war, and apparently no new drug methods had been used, with the possible exception of the administration in schizophrenia of aminopyrine, which was scarce at the time of this visit.

State Bacteriologic Laboratory

The State Bacteriologic Laboratory, Dr. Willi Rimpau, director, in normal times performed the general bacteriologic work required by the area bounded by the Danube River, Lake Constance, Ulm and Berchtesgaden. It had four medical bacteriologists, one of them the director, some thirteen technical assistants, eight typists and nine clerks to carry on the work. The institute has been largely destroyed by bombing, however, and Dr. Rimpau is at present trying to start again on two floors of a former S. A. home, but this will require official sanction and funds. If he can get started he has about six months supply of material with which to work. He also mentioned the desirability of setting up some clinical chemical assistance for clinicians.

Bavarian Institute for Occupational Medicine

The Bavarian Institute for Occupational Medicine at Munich was completely gutted and its director, Dr. Franz Koelsch, could not be traced. The address left at the old institute was now a military headquarters.

BRAZIL

(From Our Regular Correspondent)

São Paulo, Sept. 25, 1945.

Treatment of Psychopathic Patients

The Servico de Assistência a Psicopatas has undergone gratifying development during the administration of Dr. Fernando Costa in the state de São Paulo. Special attention has been given to the treatment and prevention of mental sickness. There are 8,755 patients in the hospitals of the Assistência, of whom 6,535 have entered during the administration of Dr. Fernando Costa. One of the first acts of the government was to enlarge the clinics of the Assistência, with the employment of twenty-two more doctors, some dentists and other assistants. A new hospital has been opened in Ribeirão Preto, with 400 patients and room for 1,000 beds. To attend to the patients in the rural districts Dr. Fernando Costa gave to the Assistência money to construct two outlying hospitals, one in the region served by the Central Railway and the other in the region served by the Sorocabana Railway.

Hospitals in Brazil

Hospital care in Brazil today can accommodate a large number of patients, although it has not attained complete development. In 1930 there were 607 hospitals in Brazil, which number had increased to 1,230 in 1943, located in different parts of the country. In 1930, with 42,729 beds, the number of patients cared for was 363,866. In the year 1940 653,263 patients were cared for. The number of hospital beds had increased to 109,338.

Personal

A ceremony in honor of Dr. Luis de Barros Lima, professor of surgery at the Medical School of Recife, state of Pernambuco, was held at the Santo Amaro Hospital, at which Dr. Monteiro de Morais, a professor at the school, described the twenty-five year medical career of Dr. Barros Lima, his numerous contributions to the medical literature and the prizes and medals awarded him for distinguished medical activities.

Dr. Julio Diez, Buenos Aires surgeon and professor at the university of that city, read three papers on gastroenterology at the auditorium of the Medical Division of the Bureau of Fisheries, Department of Agriculture, in Rio de Janeiro.

The University of São Paulo has released for publication a note about Dr. Edmundo Vasconcellos, professor of surgery, who is now at the Mayo Clinic in Rochester, Minn. Dr. Vasconcellos, who is accompanied by one of his assistants, Dr. Eugenio Mauro, has been invited by the U. S. State Department to visit, during three months, the principal universities of the United States to study the modern teaching of surgery.

Marriages

Marion Rudolph Mobley Jr., Florence, S. C., to Miss Elizabeth Louise Becker of Johnstown, Pa., August 25.

Thomas Whitehead Murrell Jr., Richmond, Va., to Miss Jane Nelson Goolrick of Fredericksburg, August 2.

Gerald J. Nemmers, La Crosse, Wis., to Miss Rosemay Ehred in Forest City, Iowa, recently.

Glenn G. Newman, Clinton, N. C., to First Lieut. Pearl Kaiser of Leesville, S. C., August 13.

Leo F. J. Wilking Jr. to Dr. Virginia Center Nichols, both of New York, September 15.

Julius Karl Neils, Portland, Ore., to Miss Gloria Mary Peck of St. Louis, August 10.

John E. Miller to Miss Emma Louise Schaefer, both of Quincy, Ill., in August.

Horace H. Long to Miss Nancy J. Nailor, both of Mechanicsburg, Pa., May 30.

Luette H. Kuhlman to Mr. Jack DeVorss, both of Toledo, Ohio, in August.

Deaths

Thomas **Benton Cooley** ✠ Detroit; University of Michigan Department of Medicine and Surgery, Ann Arbor, 1895; born in Ann Arbor, Mich., June 23, 1871; professor and head of the department of pediatrics from 1936 to 1941, when he became emeritus professor at the Wayne University College of Medicine; instructor at his alma mater from 1898 to 1900 and assistant professor of hygiene from 1903 to 1905; specialist certified by the American Board of Pediatrics, Inc.; member and past president of the American Pediatric Society, serving as a member of the governing council; charter member and past president of the American Academy of Pediatrics, serving also as executive secretary to its council for pediatric research; founder president of the Detroit Pediatric Society; past president of the Detroit Academy of Medicine; member of the American Institute of Nutrition, Society for Research in Child Development and the Society for Pediatric Research; major and assistant chief of the American Red Cross Children's Bureau in France during World War I; in 1924 awarded the Cross of Legion of Honor of France; for many years chief of the pediatric service and chairman of the staff of the Children's Hospital; on the consulting staff of the Harper, Woman's and Highland Park (Mich.) hospitals; died in the Eastern Maine General Hospital, Bangor, Maine, October 13, aged 74, of hypertensive heart disease. The clinical entity erythroblastic anemia is known as Cooley's disease.

Lee S. Huizenga, Shanghai, China; New York Homeopathic Medical College and Flower Hospital, New York, 1913; medical missionary in China for the Christian Reformed Church since 1920; served as superintendent of the mission hospital at Jukao and the new tuberculosis hospital in Shanghai after establishing a leprosarium there; at one time medical adviser to the American Mission to Lepers, carrying on leprosy investigations in the orient; in 1938 a delegate both from China and the United States to the International Leprosy Conference at Cairo, Egypt; at one time a minister in a church at Englewood, N. J., and a medical missionary to the Navajo Indians in New Mexico; died in a civilian internee camp in Chapei, China, July 16, aged 64, of peritoneal carcinoma.

George W. Mackenzie ✠ Philadelphia; Hahnemann Medical College and Hospital of Philadelphia, 1893; specialist certified by the American Board of Otolaryngology; member of the American Academy of Ophthalmology and Otolaryngology and the American Laryngological, Rhinological and Otological Society; fellow of the American College of Surgeons; past president of the Philadelphia Laryngological Society and the American Institute of Homeopathy; on the staffs of St. Luke's Hospital and the Children's Medical Center; in 1932 the University of Vienna bestowed on him a gold honor medal with a diploma of the university in recognition of his work in diseases of the eye, ear, nose and throat; died August 5, aged 73, of coronary thrombosis.

James Samuel Wolfstein, Cleveland; University of Michigan Medical School, Ann Arbor, 1922; member of the American Medical Association; member of the Academy of Medicine of Cleveland, serving as secretary and chairman of its section on obstetrics and gynecology; fellow of the American College of Surgeons; specialist certified by the American Board of Obstetrics and Gynecology; commissioned a major in the medical corps, Army of the United States, and served from September 1942 until he obtained his medical discharge in February 1944; for many years on the staff of the Mount Sinai Hospital, where he died July 27, aged 47, of carcinomatosis.

John **Herbert Bliss** ✠ Brooklyn; Columbia University College of Physicians and Surgeons, New York, 1921; interned at the Methodist Hospital from June 1921 to October 1923, when he became a fellow in surgery and the following year a first assistant in radium therapy at the Mayo Foundation, Rochester, Minn.; member of the Brooklyn Surgical Society; fellow of the American College of Surgeons and the International College of Surgeons; attending surgeon, Brooklyn Cancer Institute; chief surgeon, second surgical division, Methodist Hospital Clinic, and associate surgeon at the Methodist Hospital, where he died October 1, aged 52.

Carl Benjamin Rosenkrans, East Stroudsburg, Pa.; Medico-Chirurgical College of Philadelphia, 1911; member of the American Medical Association; county medical director; state game commissioner and Republican county chairman; a captain in the medical corps with the 322d Infantry, serving overseas during World War I; one of the founders of the General Hospital of Monroe County, where he served on the staff; physician and surgeon for the Lackawanna Railroad for many years; accidentally drowned, August 23, aged 57, when he was washed overboard from a fishing boat at Indian River Inlet, Rehoboth Beach, Del.

John Minor Blackford ✠ Seattle; University of Virginia Department of Medicine, Charlottesville, 1910; specialist certified by the American Board of Internal Medicine; member of the North Pacific Society of Internal Medicine, Association of the Resident and Ex-Resident Physicians of the Mayo Clinic and the American Gastro-Enterological Association; fellow of the American College of Physicians; served on the staff of the Mayo Clinic, Rochester, Minn., from 1911 to 1917; one of the founders of the Mason Clinic and of the Virginia Mason Hospital, of which he was chief of staff and where he died September 12, aged 58.

George James Dennis ✠ Chicago; Miami Medical College, Cincinnati, 1887; Northwestern University Medical School, Chicago, 1898; specialist certified by the American Board of Otolaryngology; emeritus member of the Illinois State Medical Society; member of the American Academy of Ophthalmology and Otolaryngology; at one time associate in laryngology at the Northwestern University Medical School; served on the staffs of the Wesley Memorial and Grant hospitals; died in the Passavant Memorial Hospital May 21, aged 79, of duodenal ulcer, generalized arteriosclerosis and myocardial infarction.

William Kieper Kistler, Philadelphia; Hahnemann Medical College and Hospital of Philadelphia, 1928; member of the American Medical Association; fellow of the American College of Surgeons; specialist certified by the American Board of Otolaryngology; served on the faculties of the Graduate School of Medicine of the University of Pennsylvania and his alma mater; on the staffs of the Delaware County Hospital, Drexel Hill, and the Hahnemann, Mount Sinai and Broad Street hospitals; visiting assistant surgeon, Abington (Pa.) Memorial Hospital; died July 27, aged 46, of heat exhaustion.

Craig Barrow, Savannah, Ga.; University of Maryland School of Medicine, Baltimore, 1900; member of the American Medical Association; for many years chief surgeon of the Central of Georgia Railway and its hospital; formerly medical superintendent, vice president and president of the Georgia Infirmary; served in the medical corps of the U. S. Army during World War I; at one time alderman; served as governor and lieutenant governor of the Society of Colonial Wars in the state of Georgia; died in a local hospital August 31, aged 69, of cerebral hemorrhage.

George Hursh Ensminger, Chicago; Medico-Chirurgical College of Philadelphia, 1905; clinical instructor in otorhinolaryngology at the Loyola University School of Medicine; served as health officer of Glen Ellyn and as medical consultant for several railroad companies; junior attending ophthalmologist and rhino-otolaryngologist at the Mercy Hospital, where he had been chief of the eye clinic from 1918 to 1922; assistant to the chief aurist at the Illinois Central Hospital, where he died August 23, aged 67, of hypertensive heart disease and glomerulonephritis.

Walter Baumgarten ✠ St. Louis; Washington University School of Medicine, St. Louis, 1896; served on the faculty of his alma mater; formerly secretary of the Missouri State Medical Association and editor of its journal; specialist certified by the American Board of Internal Medicine; fellow of the American College of Physicians; formerly associated with the U. S. Public Health Service; on the staffs of the Barnes, St. Luke's and Jewish hospitals; burned to death, August 23, aged 72, when his summer home near Fish Creek, Wis., caught fire.

William Douglas Anderson, Tarpon Springs, Fla.; University of Georgia Medical Department, Augusta, 1924; member of the American Medical Association; on the staff of the Morton F. Plant Hospital in Clearwater; died in Wilmington, Del., August 15, aged 51, of bleeding duodenal ulcer.

Richard Wortman Baker, Pasadena, Calif.; Beaumont Hospital Medical College, St. Louis, 1893; member of the American Medical Association; died June 7, aged 81, of arteriosclerosis.

John Joseph Bartley, Lawrence, Mass.; Harvard Medical School, Boston, 1901; member of the American Medical Association; formerly city physician; supervisor of school physicians for the city of Lawrence; died June 16, aged 73, of cerebral hemorrhage.

James William Baxter Sr. ✦ New Albany, Ind.; Kentucky School of Medicine, Louisville, 1903; past president of the Floyd County Medical Society; served in the medical corps of the U. S. Army during World War I; on the staff of St. Edward's Hospital; died August 28, aged 72, of cerebral hemorrhage and encephalitis.

Alvin Benton Caldwell, Forrest City, Ark.; Memphis (Tenn.) Hospital Medical College, 1911; member of the American Medical Association; county health officer; died in the Baptist Memorial Hospital, Memphis, Tenn., July 27, aged 66, of coronary embolism.

Georgina Helme Carruthers, Philadelphia; Woman's Medical College of Pennsylvania, Philadelphia, 1900; member of the American Medical Association; died August 23, aged 84, of senility.

Dana Lawrence Cavendish, Fayetteville, W. Va.; Medical College of Virginia, Richmond, 1927; served during World War I; a captain in the medical corps, Army of the United States, from May to August 1942, when he was released; died August 3, aged 47, of heart disease.

Joel Toy Curry, Macon, Ga.; Columbian University Medical Department, Washington, D. C., 1897; formerly chief medical officer, retirement division, U. S. Civil Service Commission in Washington, D. C.; died in Lakemont June 30, aged 73, of coronary occlusion.

William David Davis, East Canton, Ohio; Tennessee Medical College, Knoxville, 1893; member of the American Medical Association; for many years president of the board of education; formerly on the staff of the Aultman Hospital, Canton; died August 22, aged 77, of arteriosclerosis.

Louis Alexander Denis, Red Bank, N. J.; University of Pennsylvania Department of Medicine, Philadelphia, 1894; died August 2, aged 72, of coronary occlusion.

George Stephen de Renyi, Philadelphia; Magyar Királyi Pázmány Petrus Tudományegyetem Orvosi Fakultasa, Budapest, Hungary, 1916; associate professor of anatomy at the University of Pennsylvania School of Medicine and the Medico-Chirurgical College, Graduate School of Medicine, University of Pennsylvania; member of the American Association of Anatomists; died in the University Hospital August 24, aged 53, of cirrhosis of the liver.

John M. Fawcett, Coral Gables, Fla.; Pulte Medical College, Homeopathic, Cincinnati, 1889; died in the Ohio Valley General Hospital, Wheeling, W. Va., August 5, aged 81, of carcinoma of the gallbladder.

George Edwards Fay ✦ Detroit; University of Michigan Department of Medicine and Surgery, Ann Arbor, 1901; at one time affiliated with the Standard Life and Accident Insurance Company; served during World War I; died in Grosse Pointe August 23, aged 70, of heart disease.

John McKelvey Foster, Birmingham, Ala.; Memphis (Tenn.) Hospital Medical College, 1910; member of the American Medical Association; physician for the juvenile court; died in the Jefferson Hospital August 9, aged 65, of coronary occlusion.

Bernard S. Gostin ✦ Macon, Ga.; College of Physicians and Surgeons, Baltimore, 1903; on the staff of the Macon Hospital; died August 28, aged 65, of coronary occlusion.

Michael Anthony Griffin ✦ Chicago; Rush Medical College, Chicago, 1896; served on the staffs of the Columbus, St. Joseph and American hospitals; died August 23, aged 75, of chronic myocarditis.

John M. Higgins, Athens, Ohio; Pulte Medical College, Homeopathic, Cincinnati, 1893; health commissioner of Athens for many years; on the staff of the Sheltering Arms Hospital; died June 30, aged 83, of heart disease.

Francis Marion Hiller, Carterville, Ill.; St. Louis College of Physicians and Surgeons, 1909; member of the American Medical Association; on the staff of the Holden Hospital, Carbondale, where he died August 31, aged 66, of coronary occlusion.

John Henry Hovenden ✦ Laurens, Iowa; College of Physicians and Surgeons of Chicago, School of Medicine of the University of Illinois, 1898; died August 28, aged 71, of coronary thrombosis.

Nathan Louis Jacobson, Lynn, Mass.; Maryland Medical College, Baltimore, 1910; member of the American Medical Association; on the staffs of the Lynn and Union hospitals; died in the Beth Israel Hospital, Boston, July 13, aged 62, of pulmonary embolism.

William Kilburn Jaques, Chicago; Chicago Medical College, 1887; died August 6, aged 86, of cerebral hemorrhage.

Thomas Gotthart Jenny ✦ Pittsburgh; Western Pennsylvania Medical College, Pittsburgh, 1907; died in Miami Beach, Fla., August 31, aged 59, of coronary heart disease and nephritis.

Alice Elizabeth Johnson ✦ Philadelphia; Woman's Medical College of Pennsylvania, Philadelphia, 1905; member of the American Psychiatric Association; for many years clinical professor of psychiatry at her alma mater; formerly a druggist; at one time resident physician at the Pennsylvania Hospital, Department for Mental and Nervous Diseases; since 1920 psychiatrist at the Municipal Court; died in the Woman's Hospital July 19, aged 72, of cerebral thrombosis.

Forrest A. Kelley ✦ Winfield, Kan.; John A. Creighton Medical College, Omaha, 1906; for many years county health officer; on the staffs of the William Newton Memorial and St. Mary's hospitals; died July 14, aged 67, of coronary thrombosis.

Robert Oliver Kevin, Philadelphia; Jefferson Medical College of Philadelphia, 1882; for many years on the staff of the Jefferson Hospital; died July 30, aged 90, of acute dilatation of heart and arteriosclerosis.

George L. Lanius, Beaumont, Texas (licensed in Texas under the Act of 1907); died in St. Therese Hospital July 8, aged 78, of diabetes mellitus and acute nephritis.

Gaspar Emmanuel Leontine, Boston; College of Physicians and Surgeons, Boston, 1917; died June 10, aged 72, of coronary thrombosis.

Brooklyn Boyer Levengood, Bellwood, Pa.; Jefferson Medical College of Philadelphia, 1889; served overseas during World War I; in 1940 was presented with a fifty year testimonial certificate at a meeting of the Sixth Councilor District of the Medical Society of the State of Pennsylvania; on the staffs of the Mercy Hospital, Altoona, and the Nason Hospital, Roaring Springs; died July 6, aged 77, of cerebral hemorrhage.

Aaron Yancy Linville, Winston-Salem, N. C.; University of the City of New York Medical Department, New York, 1889; member of the American Medical Association; honorary member of the Medical Society of the State of North Carolina; died July 7, aged 82, of heart disease.

William Alex MacLeod ✦ New York; University of the City of New York Medical Department, 1896; died in the Yarmouth Hospital, Yarmouth, N. S., August 5, aged 76.

Joseph Elmer Magee ✦ Carnegie, Pa.; University of Pittsburgh School of Medicine, 1914; formerly Burgess of the borough of Carnegie; died August 2, aged 54, of brain tumor.

Percy Edwin Dunlop Malcolm ✦ New York; Bellevue Hospital Medical College, New York, 1893; an Affiliate Fellow of the American Medical Association; member of the American Laryngological, Rhinological and Otological Society; fellow of the New York Academy of Medicine; specialist certified by the American Board of Otolaryngology; on the staffs of the Manhattan Eye, Ear and Throat and the Metropolitan hospitals; died in Mont Tremblant Lodge, Que., Canada, August 12, aged 75.

Benjamin Lake Noyes ✦ Stonington, Maine; Medical School of Maine, Portland, 1895; died in the Eastern Maine General Hospital, Bangor, October 16, aged 75, of pneumonia.

Charles E. O'Bryan, Mortons Gap, Ky.; University of Tennessee Medical Department, Nashville, 1899; member of the American Medical Association; died August 8, aged 77, of heart disease.

William John St. Clair O'Callaghan, Nashville, Tenn.; University of Tennessee Medical Department, Nashville, 1904; served as postmaster and postal official for many years; past president and for many years member of the board of directors of the Y. M. C. A.; died July 14, aged 77, of coronary thrombosis.

Sarah Ellen Palmer, Boston; Woman's Medical College of Pennsylvania, Philadelphia, 1880; member of the American Medical Association; fellow of the American College of Surgeons; served on the staff of the New England Hospital for Women and Children; died August 23, aged 89, of myocardial failure and paralysis agitans.

Ernst J. Panetti, Milwaukee; Milwaukee Medical College, 1901; member of the American Medical Association; died August 24, aged 79, of coronary thrombosis and arteriosclerosis.

William H. Parsons, Newcastle, Maine; University of Vermont College of Medicine, Burlington, 1885; member of the American Medical Association; honorary member of the Maine Medical Association; trustee of the Miles Memorial Hospital in Damariscotta; died June 8, aged 84, of angina pectoris.

Alice Maria Patterson, Marblehead, Mass.; Boston University School of Medicine, 1893; Tufts College Medical School, Boston, 1904; member of the American Medical Association and the New England Society of Psychiatry; served as senior physician for the Wrentham State School in Wrentham; died in Danvers June 20, aged 75, of carcinoma of the pancreas.

Paul Jacob Pauliny, Tucson, Ariz.; Deutsche Universität Medizinische Fakultät, Prague, Czechoslovakia, 1927; on the staff of the San Xavier Indian Sanatorium, where he died July 22, aged 43, of hypertension, nephritis and congestive heart disease.

Richard Kirtley Pemberton ✦ McAlester, Okla.; Missouri Medical College, St. Louis, 1895; served two terms as mayor of the city of McAlester; active member of the staffs of St. Mary's and Albert Pike hospitals; died September 24, aged 74, of coronary occlusion.

James McIlvaine Phillips, Galloway, Ohio; University of Pennsylvania Department of Medicine, Philadelphia, 1898; at one time professor of pathology at the Ohio State University College of Medicine; died in the Grant Hospital, Columbus, July 28, aged 71, of shock following a prostatectomy.

Chester Arthur Poe ✦ St. Louis; St. Louis College of Physicians and Surgeons, 1914; at one time coroner of Cape Girardeau County; on the staff of the Missouri Baptist Hospital, where he died August 3, aged 58, of cerebral hemorrhage.

Philip Henry Quick, Olivet, Mich.; University of Michigan Department of Medicine and Surgery, Ann Arbor, 1895; past president of the Eat'n County Medical Society; member of the American Medical Association; past president of the local school board; formerly bank president; died in Battle Creek August 11, aged 78, of cerebral hemorrhage.

Edwin V. Ross, Rochester, N. Y.; Homeopathic Hospital College, Cleveland, 1890; at one time associated with the city health department; died August 6, aged 78, of carcinoma of the face.

Edward Sturgis Rutledge, Johnstown, Ohio; Ohio Medical University, Columbus, 1897; died July 18, aged 82, of cerebral hemorrhage.

Dorsy McNeelas Ryan, Charleston, W. Va.; Medical College of Virginia, Richmond, 1906; member of the American Medical Association; served in the medical corps of the U. S. Army during World War I; died September 19, aged 67, of acute myocardial insufficiency.

Guy W. Seaton ✦ Indianapolis; Medical College of Indiana, Indianapolis, 1898; associate in otolaryngology, Indiana University School of Medicine; member of the American Academy of Ophthalmology and Otolaryngology; specialist certified by the American Board of Otolaryngology; died in the Methodist Hospital August 12, aged 61, of coronary occlusion.

Albert Newton Seidel, Reading, Pa.; Baltimore Medical College, 1891; died in St. Joseph Hospital June 28, aged 78, of coronary thrombosis and influenzal pneumonia.

Daniel Walter Sheek, Greenwood, Ind.; Medical College of Indiana, Indianapolis, 1903; member of the board of health; served as coroner; died in St. Vincent's Hospital, Indianapolis, August 28, aged 73, of coronary occlusion.

Bradford Wyckoff Sherwood, Syracuse, N. Y.; Hahnemann Medical College and Hospital of Philadelphia, 1890; died August 29, aged 86, of senility.

John Shirk Simons ✦ Marietta, Pa.; Jefferson Medical College of Philadelphia, 1923; served on the staff of St. Joseph's Hospital in Lancaster; died July 20, aged 63, of coronary occlusion.

Benjamin L. Smith, Forsyth, Ga.; Georgia College of Eclectic Medicine and Surgery, Atlanta, 1906; member of the American Medical Association; died in the Mercy Hospital, Macon, July 7, aged 62, of angina pectoris.

John Morrison Sokol ✦ Spencer, Iowa; Rush Medical College, Chicago, 1902; for many years county coroner; died July 22, aged 69, of coronary thrombosis.

Thomas Alvin Son, Bonne Terre, Mo.; American Medical College, St. Louis, 1899; for many years a member of the state board of health; died in Kirkwood July 11, aged 88, of nephritis and fracture of the left hip received in a fall.

John Murdock Stewart, Marion Center, Pa.; University of Pittsburgh School of Medicine, 1908; served during World war I; died in the Veterans Administration Facility, Aspinwall, June 29, aged 67, of coronary heart disease.

Albert Edward Snow, St. Louis; American Medical College, St. Louis, 1893; died August 13, aged 75, of arteriosclerosis.

Charles De Witt Voorhees, Clearwater, Fla.; University of the City of New York Medical Department, New York, 1889; member of the American Medical Association and the Medical Society of the State of Pennsylvania; died July 22, aged 88, of congestive heart disease.

Lucy Nye White, Freetown, Mass.; Illinois Medical College, Chicago, 1902; died June 1, aged 84, of cerebral hemorrhage.

William L. Wilson, Scipio, Ind.; Beach Medical College, Indianapolis, 1886; member of the American Medical Association; died August 28, aged 85, of carcinoma of the prostate.

Edward Archer Wight, Bolsters Mills, Maine; Medical School of Maine, Portland, 1889; died in Wakefield July 23, aged 81, of heart disease.

PUBLIC HEALTH SERVICE

Robert Olesen ✦ Medical Director, U. S. Public Health Service, Staten Island, N. Y.; Georgetown University School of Medicine, Washington, D. C., 1905; born in New Ulm, Minn., May 22, 1883; served an eighteen month internship in Providence Hospital, Washington, D. C.; on April 1, 1908 commissioned an assistant surgeon in the regular public health service; from 1909 to 1913 served as quarantine and immigration officer in Manila, Mariveles and Iloila, serving in the last as district health officer in cooperation with the Insular Board of Health; on completion of a course at the Hygiene Laboratory, Washington, D. C., in 1913 assigned to study industrial health conditions for the U. S. Commission on Industrial Regulations in Minnesota, Michigan, Illinois and Ohio; during World War I conducted industrial surveys in New York, New Jersey, Pennsylvania, Illinois and Wisconsin for the newly created office of industrial hygiene and sanitation; served as medical director of extracantonment areas in Kentucky and Alabama; credited with organizing the bureau of communicable disease control, Wisconsin State Board of Health, in 1919 and the North Dakota State Department of Health in 1922; from 1923 to 1929, while in charge of the Office of Goiter Studies, with headquarters in Cincinnati, organized the large scale goiter surveys in various parts of the country; in 1930 assigned to the American embassy in Berlin, where he was in charge of physical and mental examinations of emigrants to the United States; returned to the United States in 1934 and assigned to the Farm Security Administration, for whose clients he developed a public health and medical program; in April 1936 assigned as assistant surgeon general in charge of the division of sanitary reports and statistics, U. S. Public Health Service; in 1939 assigned to the Foreign Quarantine Station at Rosebank; became a passed assistant surgeon in the U. S. Public Health Service in July 1912, surgeon in June 1920, senior surgeon in July 1930 and medical director in March 1934; died at his home in the quarantine station, Rosebank, August 15, aged 62, of cerebral hemorrhage.

Bruno Solby ✦ Surgeon, U. S. Public Health Service (R), Federal Security Agency, Washington, D. C., Medizinische Fakultät der Universität Wien, Vienna, Austria, 1932; member of the Society of American Bacteriologists, American Sociological Society and the Medical Society of the State of New York; associate member of the American Psychiatric Association; specialist certified by the American Board of Psychiatry and Neurology, Inc.; took postgraduate work in neuropathology, neurology and psychiatry at the New York Post-Graduate Hospital, where he had been assistant neurologist and psychiatrist; a captain in the medical corps, Army of the United States, from August 1942 to October 1943, when he was honorably discharged; joined the public health service in November 1943; detailed to the U. S. Public Health Service Dispensary, Washington, where he died September 8, aged 41, of cerebral hemorrhage.

Henry S. Wolanczyk, Assistant Surgeon, U. S. Public Health Service Reserve, Buffalo; University of Buffalo School of Medicine, 1940; diplomate of the National Board of Medical Examiners; interned at the U. S. Marine Hospital in San Francisco; joined the public health service reserve on July 1, 1941; served as public health officer for the Laclede County Health Department in Lebanon, Mo.; assigned to Coast Guard aboard the U. S. S. *Harveson* Oct. 1, 1943; died in the U. S. Marine Hospital, Portsmouth, Va., Dec. 23, 1943, aged 28.

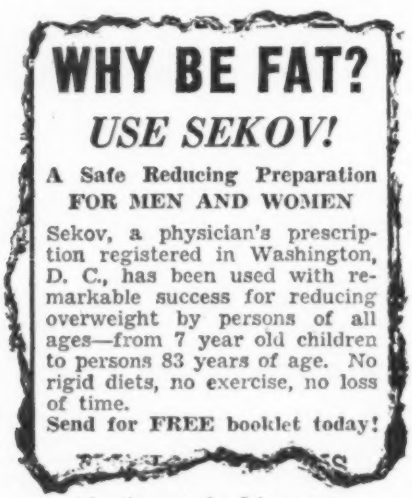
Bureau of Investigation

SEKOV DANGEROUS TO HEALTH

Federal Court Forbids Further Interstate Sale of "Reducer"

"Home treatments" for obesity frequently contain thyroid, with all its dangerous side effects. Disastrous results from self dosing with these preparations are common. Certain such "cures" have persisted in the market for years in spite of the government's efforts to protect the public by checking their sale. The perennial "Marmola" is a conspicuous example.

Another such nostrum—"Sekov"—has thrived for at least ten years, although actions against its promoters have been initiated by two federal agencies. Sekov is the reverse spelling for "Vokes," the name of its backers, Edwin H. and Hazel R. Vokes of Hollywood, Calif., whose latest trade styles appear to be "Sekov Studios" and "Sekov Corporation." An investigation made a few years ago indicated that Hazel Vokes got her start in the weight-reducing business by working for Gertrude Nova, Inc., of Hollywood, apparently a trade style for a woman who was sometimes known variously as Gertrude Nova and Gertrude Tenderich, and who was once in a similar


WHY BE FAT?
USE SEKOV!
A Safe Reducing Preparation
FOR MEN AND WOMEN
Sekov, a physician's prescription registered in Washington, D. C., has been used with remarkable success for reducing overweight by persons of all ages—from 7 year old children to persons 83 years of age. No rigid diets, no exercise, no loss of time.
Send for FREE booklet today!

Advertisement of a Sekov agent.

business in Denver called the Avon Laboratories (note that "Avon" is the reverse spelling of "Nova"). Apparently this person sold out her Denver concern and established a similar one in Hollywood, reportedly at the same address as that later used by the Vokes-Sekov outfit. Hazel Vokes is said to have bought the Nova business in 1935. Presumably the formula of the Nova nostrum—long alleged to contain thyroid—was included in the reported sale of her business to Hazel Vokes.

In August 1936 the Los Angeles Health Department reported that when it was investigating Sekov an agent supplied two "formulas" for it, as follows:

No. 1. Thyroid 1½ gr.
Big pill whole Pit. 1 gr.
Big pill whole Ovar. 3 gr.
R ½ is ½ of No. 1. Digitalis ½ gr.

No. 2. Rhubarb root 2 gr. Cascara Sagrada 1 gr.
Aloin 1/16 gr. Asafetida ½ gr.
Oleoresin ginger 1/16 gr.
T. ½ size. Digitalis ½ gr. Thyroid 2 gr.
P. ½ size. Whole Pit. 2 gr. Thyroid 1 gr.
O. ½ size. 2 gr. Ova. whole. 1 gr. whole Pit.

The presence of thyroid in these nostrums was later confirmed by chemists of the Food and Drug Administration. According to their report, each carton of the nostrum contained two types of capsules, "No. 1" and "No. 2." The No. 1, they said, consisted of glandular material, including thyroid in amounts ranging from 1.84 to 1.95 grains per capsule, the No. 2 rhubarb root, cascara sagrada bark, aloin and asafetida, and the "Formula P" approximately 1.72 grains of thyroid per capsule.

The first government agency to take action against the Vokes persons was the Federal Trade Commission, which, on Sept. 26, 1940 ordered them to discontinue any of their advertisements which did not warn that the use of Sekov under the conditions prescribed in the advertising, or those customary or usual, might result in serious or even irreparable injury to health. The Commission's findings included the charge that even when Sekov might produce a reduction in weight by accelerating the rate of metabolism it might seriously weaken the body and its organs, including the heart, by means of the thyroid present. The details of this case were given in THE JOURNAL, Aug. 9, 1941, page 472.

In March 1945 the Food and Drug Administration published the results of two actions that it had brought against the Sekov outfit following the seizure of interstate shipments of Sekov preparations. The seizures were based on the charge that the articles were misbranded under the Food, Drug and Cosmetic Act in that the labeling was false and misleading, that the nostrums would be dangerous to health when taken in the dosage or with the frequency or duration recommended on the labels, and that the latter failed to give adequate directions for use and such warnings as are necessary in the case of thyroid and laxative preparations. One product, the "Formula P," was also declared to be adulterated in that each tablet contained more than the 1 grain of thyroid represented. These findings were confirmed by a district federal court in California despite the protests of the Sekov promoters, and the charge of misbranding was sustained under Notice of Judgement No. 1002, issued in March 1945.

In this connection, Notice of Judgment No. 1001 reported that, in view of the danger attending the use of the Sekov nostrums, the court issued an injunction forbidding the Vokeses thereafter to ship any of their products known as "Sekov," "Sekov Reducer," "Sekov Reducer for Men" and "Sekov Formulas P, R and T" in interstate commerce, which action means that they cannot be shipped out of California because they are potentially dangerous to health.

Thus the public outside of California is to be protected from the risk involved in taking Sekov. Whether the California officials will be willing to "harm home industry" by restricting the sale of Sekov in its own state, or simply follow the "caveat emptor" policy, remains to be seen.

SOME MISCELLANEOUS MEDICAL FRAUDS

A Variety of Schemes Debarred from the Mails

Fraud orders issued by the Post Office Department have frequently been the subject of extensive articles by the Bureau of Investigation in these pages of THE JOURNAL. Following is a brief abstract of one such fraud order:

Perma Company and Twil-Gro Company.—These were trade styles used by one Ollie Altman of Brooklyn in selling through the mails a product known as "Perma Youthskin" or "Perma Smoothskin." This, he represented, would remove wrinkles and crows'-feet, benefit scrawny necks and baggy eyelids, restore the texture of the skin and the appearance of the user to their former youthful condition, regardless of the age of the user. After the Post Office Department had investigated the whole business, it notified Altman to appear at a hearing in Washington and answer to a charge of using the mails to defraud. He did not appear, but sent counsel. A government chemist testified that his analysis of Perma Youthskin disclosed 35 per cent of paraffin and spermaceti, about 29 per cent of lanolin, nearly 20 per cent of water, more than 10 per cent of beeswax, 5 per cent of turtle oil, less than 1 per cent of borax, and a trace of perfume. A Senior Medical Officer of the Food and Drug Administration also testified for the government, showing that wrinkles are a natural accompaniment of age, but may result from some other causes, such as sudden and considerable loss of weight, undernourishment, improper functioning of certain endocrine glands, and constant exposure to the glare of the sun. He discussed the structure of the skin and its tissues, and showed the futility of attempting to correct deep wrinkles by a combination of turtle oil and lanolin, which ingredients the promoters of Perma Youthskin particularly emphasized. He testified, further, that turtle oil has no more beneficial effects on the skin than any other animal oil, and that the preparation in question would benefit the skin no more than any other grease applied to it with massage. He pointed out that the product did not supply or build up the missing tissues, though it might cause some of the fine lines in the face to be temporarily less apparent. Even the unnamed physician who testified for the defendant admitted the foregoing statements about turtle oil and agreed with the government testimony that baggy eyelids resulting from systemic diseases could be removed only by treatment and cure of such diseases. On May 4, 1944, a fraud order was issued to debar from the mails the business operating under the names Perma Company and Twil-Gro Company, and their officers and agents.

Correspondence

DEATH FROM AIR EMBOLISM FOLLOWING INSUFFLATION DURING PREGNANCY

To the Editor:—In the article by Dr. Herbert S. Breyfogle entitled "Death from Air Embolism Following Insufflation During Pregnancy" (THE JOURNAL, September 29, p. 342) the statement is made that "the following death from air embolism resulting from insufflation during pregnancy is therefore the fourth from this means to be reported, and the first to be recorded in the medical literature of this country."

My interest was aroused, since I had reported two deaths from air embolism due to the use of vaginal powder insufflators (one during pregnancy) in the May 1945 issue of the *American Journal of Surgery*. This was approximately four months before the publication of Breyfogle's paper. Therefore, although I hate to enter into discussion about such small issues, it seemed that there might be some question as to the accuracy of the statement quoted. This author, evidently after reading my publication in May and apparently without investigation, added the following reference to his article:

> 14. Since this article was submitted for publication an additional death from air embolism following insufflation during pregnancy has been reported (Martland, H. S.: Air Embolism: Fatal Air Embolism Due to Powder Insufflators Used in Gynecologic Treatments, Am. J. Surg. **130**: 164 [May] 1945).

Investigation at the office of THE JOURNAL indicates that Breyfogle's article "was submitted to THE JOURNAL under date of Feb. 23, 1945," whereas the editor of the *American Journal of Surgery* informs me that my article was submitted Jan. 29, 1945. Therefore if we are to follow the established custom that "priority is usually calculated from the date when the article is received in the office of publication," the statement by Breyfogle that his case was "the first to be recorded in the medical literature of this country" is quite inaccurate.

However, except "to keep the record straight," priority is apparently of little significance in the report of this type of case. For it is undoubtedly true that similar catastrophes have occurred prior to the cases reported, but because of medicolegal reasons (civil suits, criminal liability and the like) have remained unreported. This is strongly supported by the experience of medical examiners in the Metropolitan Area of New York, who not infrequently investigate cases of death due to air embolism.

These deaths usually follow criminal abortions, especially by the syringe method, and wounds of and operations on the neck, upper thorax or head which involve the dural sinuses. Less frequently are encountered deaths following diagnostic and therapeutic uterine injections, transuterine pneumoperitoneum, lavage of the nasal sinuses, especially the antrum of Highmore, aerograms of the bladder, placenta previa and air injection in the region of the adrenal glands for roentgenographic visualization (Martland, H. S.: Air Embolism, with Special Reference to Its Surgical Importance, *Am. J. Surg.* 68:281 [June] 1945).

HARRISON S. MARTLAND, M.D., Newark, N. J.
Chief Medical Examiner of Essex County.

To the Editor:—Dr. Breyfogle's report of a death from air embolism following insufflation during pregnancy (THE JOURNAL, September 29, p. 342) interested me deeply. Contrary to Dr. Breyfogle's mention of the first reference in medical literature to insufflation into the vagina in 1936, I have to say that the dry treatment of discharge of different kinds by insufflation was used in Europe much earlier. I myself published about a hundred cases dealing with this in December 1912 in the *Therapie der Gegenwart*, stimulated by the publication of Nassauer and Abraham some years prior. Fortunately we had not had any deaths by insufflation because we did not use this method in pregnancy, as far as I recall.

While the aforementioned authors used the Nassauer insufflator, I preferred not to do this. I noticed in beginning of my dry treatments the defects and insufficiencies of this instrument, which are (1) frequent obstruction and therefore blowing of a great amount of air into the vagina by forceful manipulation of the physician, (2) requirement of much time to bring into the vagina the intended quantity of the powder and (3) the time needed for the changing and sterilization of the nozzle of the insufflator, which is almost impossible in a big practice or a crowded clinic.

Therefore I changed my technic in 1912. I took a piece of paper, which I folded at both sides, placing the powder (2 to 3 Gm.) in the center and inserted the speculum into the vagina. I put the powder on the speculum and applied it by means of a forceps with cotton everywhere I wanted it (to the portio and vaginal walls), which procedure was accomplished by movements of the speculum. Instead of a piece of paper I now use the glass tube in which the powder is supplied. The success of this treatment and technic was excellent and I imagine also more effective than insufflation because more powder can be brought in. Besides the dangers reported by Dr. Breyfogle can be avoided through this. There is no positive air pressure as by insufflation.

The advice given by the author to abandon insufflation in cases of pregnancy and multiparas should be considered. With the explanation of my technic of dry treatment of discharge regardless of cause, I want to make it clear that this administration of the powder avoids the dangers of the insufflator, even in cases of pregnancy and multiparas.

HEDWIG PRAGER, M.D., New York.

FOOD POISONING BY SALMONELLA MONTEVIDEO

To the Editor:—In THE JOURNAL, September 1, an outbreak of food poisoning due to Salmonella montevideo in an army general hospital is reported. That outbreak possibly was not as local as the authors believe. During the night of April 16-17, 1944 there was a similar outbreak in another hospital in southern England. As the outbreak took place a few days before that hospital started to admit patients, all cases were personnel from that hospital, approximately 250 out of 400. The cases came from both hospital messes (officers' and enlisted men's). The symptoms (abdominal cramps, watery diarrhea, nausea, high fever) were the same as described in the April 11-12 outbreak. Several patients had cough and expectoration, one with definite pneumonic signs on physical examination. The hospital's x-ray equipment had not been set up yet.

Our experience with sulfaguanidine (3 Gm. every four hours) was apparently satisfactory. The 30 patients (two wards) treated with that drug in adequate doses improved faster and were returned to duty within three and four days after the onset of symptoms. Others required from four to fourteen days of hospitalization, and the patient with frank pneumonia recovered in approximately four weeks under sulfadiazine therapy.

The organism in the stools that had been sent to the First Medical Laboratory was identified as Salmonella montevideo.

Medical officers with field units in the neighborhood told me of similar outbreaks during the same week.

Although it is possible that these outbreaks of Salmonella montevideo food poisoning were independent of one another and just happened during the same week, one has to consider the possibility of their having a common source rather than cook carriers.

NIELS L. LOW, Captain, M. C., A. U. S.

Bureau of Legal Medicine and Legislation

MEDICOLEGAL ABSTRACTS

Medical Practice Acts: Graduate of Middlesex University Not Entitled to Licensure in New Jersey.—Rothseid, a "graduate of Middlesex University School of Medicine of Massachusetts," after examination was licensed to practice medicine and surgery in Massachusetts. Thereafter he served as a resident physician in the Lawrence General Hospital, Lawrence, Mass. Subsequently he applied to the state board of medical examiners of New Jersey in the alternative either for a license by reciprocity on the basis of his Massachusetts license or to be admitted to an examination for a license. The applicable sections of the New Jersey medical practice act require an applicant for a license to practice in New Jersey to have received a diploma from a professional school which in the opinion of the board "was in good standing at the time of the issuance of the diploma." Since Middlesex University, in the opinion of the board, was not in good standing either at that time or at the time of the issuance of the diploma to Rothseid, Rothseid's application was denied by the board. Rothseid then sought a writ of mandamus either to compel the board to license him by virtue of his Massachusetts license or to admit him to examination for such a license, and the matter came before the supreme court of New Jersey.

The question to be determined here, said the supreme court, is whether the board of medical examiners is justified in classifying Middlesex University School of Medicine as an institution which was not in good standing. The board bases its conclusion in the grading and standing of medical schools on the standard adopted by the "Committee of Education" (presumably the Council on Medical Education and Hospitals) of the American Medical Association and makes public the names of the schools which it approves. Rothseid argued that the action of the board in accepting the grading of medical schools by the American Medical Association "is without using its own judgment or discretion but constitutes an unlawful delegation of its duty and power." We do not agree with Rothseid's contention, continued the court. We think the board of medical examiners is entirely within its discretion to adopt the standard and grading of an organization of which it has knowledge and confidence. We think the board acted within its sound discretion within the meaning of the medical practice act and was not actuated by arbitrariness or capriciousness. Compare *Salowitz* v. *Michigan State Board*, 285 Mich. 214, 280 N. W. 737; *Rosenthal* v. *State Bar Examining Committee*, 116 Conn. 409, 165 A. 211, 87 A. L. R. 991.

The court accordingly concluded that Rothseid had not shown any violation of his rights which entitled him to a writ of mandamus and therefore denied his application for such a writ. —*Rothseid v. State Board of Medical Examiners, 38 A. (2d) 444 (N. J., 1944).*

Hospitals: Duty to Safeguard Delirious Patient from Self Injury.—For a number of years the plaintiff had been addicted to the excessive use of liquor and on Jan. 5, 1943 voluntarily entered a sanatorium conducted by the defendant corporation "for the cure of chronic alcoholics." She was assigned a room on the second floor of the institution. When she entered the sanatorium she was highly nervous and later at times was "delusional." She did not recognize a relative who visited her. She labored under the impression that she was in an insane asylum and was discovered scrubbing the floor of her room thinking it was her kitchen floor at home. Apparently, she was subject to restraint of no nature at the sanatorium. About two days after her admittance she was found seriously injured lying on the cement pavement below the window of her room from which she had either jumped or fallen. Subsequently she instituted suit against the defendant for damages, charging that the defendant was negligent in failing to guard and restrain her and in failing properly to bar her window to prevent her plunging through the window to the ground below. At the conclusion of the plaintiff's evidence the trial court directed a verdict in favor of the defendant and entered judgment thereon. The plaintiff then appealed to the district court of appeal, second district, division 2, California.

The defendant sanatorium, said the appellate court, was under the obligation to use reasonable care in providing for and in guarding the safety of its patients, the obligation to use the care and take the precaution to prevent danger which a reasonable man would take under the circumstances shown to exist. The circumstances to be considered here included the patient's mental and physical condition and what she was likely to do because of that condition; but the defendant's obligation was subject to the rule that it was not required to use greater care than a reasonable person would exercise under the circumstances. The defendant was not required to take measures to avert injury to its patients which circumstances did not indicate to be reasonably necessary. Specifically, the defendant was not required to restrain its patient or bar its windows unless facts were brought to its attention showing that it was reasonably probable that the patient would do injury to herself or to some other person. In particular, in arriving at this conclusion, the court relied on the decision in *Papini* v. *Alexander Sanitarium, Inc.*, 12 Cal. App. (2d) 249, 55 P. (2d) 270. In that case the plaintiff sought damages resulting from the death of their son, who had been killed by a razor blade wielded by a fellow patient at the defendant hospital, one Boliva, who had been admitted as an "amnesia patient." In that case the appellate court reversed a judgment for the plaintiffs, holding that there was no evidence to show that the defendant knew that Boliva was suffering from "homicidal mania," that unless restrained he might voluntarily attack other patients, that he had attacked other persons on previous occasions or that he was a dangerous patient to be at large unrestrained. In this case the plaintiff relied on a number of cases in which awards were upheld for damages received by patients in jumping from windows or otherwise but, answered the court, in those cases facts were shown in evidence which indicated to the defendants that the patients involved were likely to inflict injury on themselves or others. No case was cited to this court in which a judgment was affirmed on proof that the patient was merely "delusional" or suffering from other mental disease, unaccompanied by acts pointing to violence. The court accordingly concluded that the evidence presented by the plaintiff in this case was insufficient to entitle her to recover judgment.

The judgment in favor of the defendant sanatorium was accordingly affirmed.—*Wood v. Samaritan Institution, Inc., 156 P. (2d) 470 (Calif., 1945).*

Medical Examinations and Licensure

COMING EXAMINATIONS AND MEETINGS

BOARDS OF MEDICAL EXAMINERS
BOARDS OF EXAMINERS IN THE BASIC SCIENCES

Examinations of the boards of medical examiners and boards of examiners in the basic sciences were published in THE JOURNAL, Nov. 17, page 825.

EXAMINING BOARDS IN SPECIALTIES

AMERICAN BOARD OF DERMATOLOGY & SYPHILOLOGY: *Written. Group B.* April 22. *Oral. Group A and B.* June 6-8. Final date for filing application is March 1. Sec., Dr. George M. Lewis, 66 E. 66th St., New York 21.

AMERICAN BOARD OF INTERNAL MEDICINE: *Written.* Various centers, Feb. 18. Final date for filing application is December 1. Asst. Sec., Dr. W. A. Werrell, 1 W. Maine St., Madison, Wis.

AMERICAN BOARD OF NEUROLOGICAL SURGERY: *Oral.* Chicago, May 27. Final date for filing application is Feb. 1. Sec., Dr. P. C. Bucy, 912 S. Wood St., Chicago 12.

AMERICAN BOARD OF OPHTHALMOLOGY: Chicago, Jan. 18-22 and Los Angeles, Jan. 28-Feb. 1. Sec., Dr. S. Judd Beach, 56 Ivie Rd., Cape Cottage, Me.

AMERICAN BOARD OF OTOLARYNGOLOGY: Spring 1946. Sec., Dr. Dean M. Lierle, University Hospital, Iowa City, Ia.

AMERICAN BOARD OF PEDIATRICS: *Oral.* Atlantic City, Dec. 7-8. Sec., Dr. C. A. Aldrich, 115½ First Ave., S.W., Rochester, Minn.

AMERICAN BOARD OF PSYCHIATRY & NEUROLOGY: *Oral.* New, Dec. 20-22. Chicago, May 24-25. Final date for filing application is February 1946. Sec., Dr. Walter Freeman, 1028 Connecticut Ave., N.W., Washington 6, D. C.

AMERICAN BOARD OF SURGERY. *Written.* Part I. Spring 1946. Final date for filing application is Dec. 1. Sec., Dr. J. Stewart Rodman, 225 South 15th St., Philadelphia.

AMERICAN BOARD OF UROLOGY: *Written.* Various centers, Dec. 9. *Oral.* Chicago, February 1946. Sec., Dr. Gilbert J. Thomas, 1409 Willow St., Minneapolis 4.

Current Medical Literature

AMERICAN

The Association library lends periodicals to members of the Association and to individual subscribers in continental United States and Canada for a period of three days. Three journals may be borrowed at a time. Periodicals are available from 1935 to date. Requests for issues of earlier date cannot be filled. Requests should be accompanied by stamps to cover postage (6 cents if one and 18 cents if three periodicals are requested). Periodicals published by the American Medical Association are not available for lending but can be supplied on purchase order. Reprints as a rule are the property of authors and can be obtained for permanent possession only from them.

Titles marked with an asterisk (*) are abstracted below.

American Journal of Hygiene, Baltimore

41:243-400 (May) 1945

Studies on Tsutsugamushi Disease (Scrub Typhus, Mite Borne Typhus) in New Guinea and Adjacent Islands: Epidemiology, Clinical Observations, and Etiology in Dobadura Area. F. G. Blake, K. F. Maxcy, J. F. Sadusk Jr., G. M. Kohls and E. J. Bell.—p. 243.
Id.: Further Observations on Epidemiology and Etiology. G. M. Kohls, C. A. Armbrust, E. N. Irons and C. B. Philip.—p. 374.

42:1-106 (July) 1945

Development of 1943 Vaccination Study of Commission on Influenza. T. Francis Jr.—p. 1.
Vaccination Against Influenza at University of Minnesota. E. R. Rickard, Minnie Thigpen and J. H. Crowley.—p. 12.
Value of Influenza Vaccination When Done at Beginning of Epidemic. W. M. Hale and A. P. McKee.—p. 21.
Vaccination Against Influenza: Study in California During Epidemic of 1943-1944. M. D. Eaton and G. Meiklejohn.—p. 28.
Human Immunity Following Vaccination with Formalinized Influenza Virus. G. K. Hirst, N. Plummer and W. F. Friedewald.—p. 45.
Clinical, Epidemiologic and Immunologic Evaluation of Vaccination Against Epidemic Influenza. J. E. Salk, W. J. Menke Jr. and T. Francis Jr.—p. 57.
Evaluation of Vaccination Against Influenza. T. P. Magill, N. Plummer, W. G. Smillie and J. Y. Sugg.—p. 94.

American Journal of Medical Sciences, Philadelphia

210:281-420 (Sept.) 1945

Adrenalin Administration in Persistent Anxiety States. D. E. Cameron.—p. 281.
*Eosinophilic Lung (Tropical Eosinophilia). P. J. Hodes and F. C. Wood.—p. 288.
Laryngeal Edema, Myocarditis and Unexpected Death (Early Acute Laryngotracheobronchitis). O. Saphir.—p. 296.
Life Cycle of Erythrocyte After Splenectomy and Problems of Splenic Hemolysis and Target Cell Formation. K. Singer and L. Weisz.—p. 301.
General Acquired Anhydrosis. H. T. Engelhardt and J. P. Melvin Jr.—p. 323.
Poisoning by Hydroquinone and Mono-Methyl-Paraminophenol Sulfate: Report of 2 Cases with Autopsy Findings. I. Zeidman and R. Deutl.—p. 328.
Xiphosternal Crunch and Its Incidence in Healthy Inductees. M. Solis-Cohen.—p. 333.
Heredofamilial Neurologic Disease, Resembling Charcot-Marie-Tooth Type of Progressive Muscular Atrophy, in Chinese Family. H. S. Gaskill.—p. 342.
Study of the Goitrogen Promizole with Reference to Thyroid, Metabolism and Blood. G. M. Higgins.—p. 347.
Interference Between Inactive and Active Viruses of Influenza: III. Cross Interference Between Various Related and Unrelated Viruses. W. Henle and Gertrude Henle.—p. 362.
Id.: IV. Nature of Interfering Agent. Gertrude Henle and W. Henle.—p. 369.
*Methionine in Treatment of Toxic Hepatitis. J. H. Eddy Jr.—p. 374.

Eosinophilic Lung.—According to Hodes and Wood, so-called eosinophilic lung is ushered in by lassitude, loss of appetite and fever. During the second week of illness the patient usually develops a dry hacking cough, which is worse at night. The cough becomes more severe and paroxysmal, eventually interfering with sleep. Wheezing and expiratory dyspnea develop. Many patients have symptoms of asthmatic bronchitis. Some have severe paroxysms of asthma requiring epinephrine. In rare instances an explosive asthmatic attack may be the first sign of the patient's illness. After several weeks the fever subsides, weakness gradually disappears and loss of weight ceases. The bronchopulmonary symptoms usually persist and become chronic if treatment is not instituted. Physical examination reveals hyperresonance of the chest. Sibilant and sonorous ronchi usually are present, with occasional crepitant rales at the bases. The spleen is moderately enlarged in about half of the cases. The most striking feature of eosinophilic lung is the massive eosinophilia. Roentgenograms of the chest made at the end of the second week of the febrile period reveal mottling throughout both lungs. The authors report 2 cases. They find that five injections of neoarsphenamine at four day intervals are effective in treatment. Without arsenical therapy the condition tends to persist.

The Xiphosternal Crunch.—Solis-Cohen says that in about one fifth of healthy men a crunching sound may be heard over the lower part of the sternum and the ensiform cartilage, accompanying one or both sounds of the heart and occurring chiefly during systole. The intensity of this sound is slight in a little less than half the cases, moderate in a little more than one third and pronounced in slightly more than one fifth. This crunch is often transmitted to the left, occasionally being heard as far as the apex. The crunch seems to have no pathologic significance. The importance of its recognition lies in its sometimes being regarded as an organic or congenital heart murmur or as a pericardial friction rub. Because of this sound men have been denied life insurance or have been rated up, have been refused employment, have been rejected by the armed forces and have been subjected to unnecessary treatment. Although this sound has been reported on a number of occasions from 1819 to the present time, it seldom is referred to in books or articles and is recognized by few clinicians.

Methionine in Hepatitis.—In the treatment of 30 cases of acute toxic hepatitis, most of which were caused by trinitrotoluene, Eddy employed methionine without any fatalities. At least 10 of the patients were seriously ill. This is of interest in view of the published mortality in trinitrotoluene hepatitis of 30 to 35 per cent. Of the 2 patients with trinitrotoluene hepatitis seen prior to using methionine, 1 died and the second had a prolonged illness. Many of the patients since that time have appeared to be as severely poisoned as were the first 2. Among the patients with toxic hepatitis treated with methionine the length of hospitalization varied from four days to seven weeks, with an average of thirteen days. The diagnosis was based on an analysis of the occupational exposure, physical examination, icterus index, Hanger cephalin flocculation test and urinalysis for urobilinogen and bilirubin. These examinations almost always supported one another, and they gave an excellent method of follow-up. Eddy is particularly impressed with the value of a daily urinalysis for urobilinogen and bilirubin in that it has followed the course of the illness more closely than other examinations and is easily performed. Treatment consists of bed rest, a diet rich in protein and carbohydrate and poor in fat, administration of multiple vitamin products plus added vitamin B complex and the oral administration of methionine in doses of from 3 to 8 Gm. daily. Several other patients with hepatitis due to carbon tetrachloride and 2 with epidemic hepatitis are thought to have been improved by methionine.

American Review of Soviet Medicine, New York

2:484-592 (Aug.) 1945

*Tissue Therapy in Cutaneous Leishmaniasis. V. P. Filatov.—p. 484.
Agonal States and Clinical Death: Problems in Revival of Organisms. V. A. Negovski.—p. 491.
Respiration and Anaerobic Glycolysis of Livers and Hepatomas in Mice Fed Orthoaminoazotoluene. N. V. Eltsina.—p. 500.
Adaptation to Anoxia at Different Age Levels. I. A. Arshavski.—p. 508.
Relation of Wound Exudate to Healing. M. P. Pokrovskaya and M. S. Makarov.—p. 513.
Delayed Surgical Care of Complicated Gunshot Wounds. A. I. Savitski.—p. 519.
*Transplantation of Cornea. O. I. Shershevskaya.—p. 525.
Specific Titration of Anti-Measles Serum by Complement Fixation. G. A. Orlov.—p. 531.

Tissue Therapy in Cutaneous Leishmaniasis.—Filatov reports the results of tissue therapy in the form of skin grafts or aloe leaf extract on cutaneous leishmaniasis. Seven cases of cutaneous leishmaniasis are reported in which either of these two modes of tissue therapy was employed. The cases demonstrate a powerful effect tissue therapy produces in cutaneous leishmaniasis. Grafting of conserved skin and subcutaneous injections of an extract of preserved aloe leaves are equally effective.

Transplantation of Cornea.—According to Shershevskaya, of the 435 keratoplasties performed in Filatov's clinic between 1922 and 1938 success was obtained in 24.1 per cent by the use of corneas from living donors. Filatov and his school developed the use of preserved cadaver cornea. In the Odessa clinic 264 transplantations were done, using the cornea from a cadaver. The successful results in these cases amounted to 67 per cent. Filatov perfected the technic of the operation, suggested a series of new methods, constructed instruments which made this operation simpler and easier, and widely popularized keratoplasty. Her results aroused the attention of ophthalmologists. Corneal transplantation is being performed in many hospitals throughout the Soviet Union.

American Review of Tuberculosis, New York

52:89-178 (Aug.) 1945

Tuberculosis in Household Associates: Influence of Age and Relationship. Ruth R. Puffer, H. C. Stewart and R. S. Gass.—p. 89.
Tuberculosis Among Montana Indians. J. R. McGibony and A. W. Dahlstrom.—p. 104.
*Immobilization of Both Lungs: Treatment of Pulmonary Tuberculosis in Equalizing Pressure Chamber. A. L. Barach.—p. 122.
Bronchography in Pulmonary Tuberculosis: VI. Thoracoplasty. B. A. Dormer, J. Friedlander and F. J. Wiles.—p. 145.
Anatomic Studies on Human Tuberculosis: XVIII. Additional Observations on Progressive Primary Pulmonary Tuberculosis in Adults. K. Terplan.—p. 155.

Immobilization of Both Lungs.—Barach shows that, whereas in the Thunberg type of barospirator the patient could not entirely dispense with voluntary respiration, in his apparatus this is possible. The same alternating pressure wave of 110 mm. of mercury was produced, but a partition between the head and chest end of the chamber made possible a simultaneous arrival of an equal pressure on the two surfaces of the chest wall. The arterial oxygen saturation and the carbon dioxide content were found to be normal even in patients with advanced pulmonary tuberculosis in the absence of all voluntary respiration. The ability to dispense with the recurrent impulse to breathe required training which was achieved in 1 patient in a period of forty-five minutes and in the majority of patients in three to four hours. In 10 patients who have been treated for four months and in 5 given several courses of four months each, arrest of lung movement has been obtained in all. In 4 of these patients either little improvement took place or the benefit obtained was temporary. In the remaining 6 patients significant improvement was observed. In 1 patient collapse of a large cavity took place in four months, recurred on slight activity and was closed again in a second course with fibrous tissue scar formation. Subsequently a cavity 1 cm. in diameter was seen by x-ray at the original site. Repeated individual and concentrated sputum tests have been negative for two years. The remaining 5 patients became clinically well and were discharged as able to work. Four of them are known to be at work, free from evidence of illness, with negative sputums and either without signs of tuberculosis by x-ray or with fibrotic arrest of the disease.

Archives of Neurology and Psychiatry, Chicago

54:75-162 (Aug.) 1945

Progressive Facial Hemiatrophy. R. Wartenberg.—p. 75.
Localizing Value of Temporal Crescent Defects in Visual Fields. H. A. Shenkin and I. H. Leopold.—p. 97.
*Degeneration of Peripheral Nerves in Pernicious Anemia. D. B. Foster.—p. 102.
Emotions and Adrenergic and Cholinergic Changes in Blood. O. Diethelm, E. J. Doty and A. T. Milhorat.—p. 110.
Effects of Transient Stretching of Peripheral Nerve. D. Denny-Brown and Margaret M. Doherty.—p. 116.
Central Nervous System in Uremia: Clinicopathologic Study. J. Knutson and A. B. Baker.—p. 130.
Electroencephalogram of Dogs with Experimental Space-Occupying Intracranial Lesions. G. Ulett.—p. 141.

Peripheral Nerves in Pernicious Anemia.—Foster describes pathologic changes in the peripheral nerves in 4 cases of pernicious anemia in which necropsy was performed and the results of biopsy of a peripheral nerve in an additional case. Three cases in the present series are examples of untreated pernicious anemia of many years' duration in severe hematologic relapse at the time of pathologic examination. In all there was evidence of degeneration of the peripheral nerves, manifested by reduction in myelin sheaths and axis cylinders, degeneration of myelin, increase in Schwann cells and endoneurial connective tissue, axonal reaction changes in the ganglion cells of the posterior root ganglions and degenerative changes in the intramedullary course of afferent posterior root fibers. The other 2 patients exemplify treated pernicious anemia, with the patients in hematologic remission at the time of death. In these 2 patients, who had received liver therapy, the changes were less severe. The author concludes that the great regenerative capacity of peripheral nerves offers an anatomic explanation for the clinically observed improvement with liver therapy in some of the neurologic manifestations of pernicious anemia.

Archives of Ophthalmology, Chicago

34:83-180 (Aug.) 1945

Lability of Ocular Tension: Test to Determine Individual Variations. S. Bloomfield and R. K. Lambert.—p. 83.
*Gonorrheal Syndrome without Gonorrhea: Reiter's Disease. R. L. Lucas and H. Weiss.—p. 97.
Local Toxic Effect of Detergents on Ocular Structures. I. H. Leopold.—p 99.
Plastic Surgical Repair About Eyes with Free Grafts. A. Schultz and C. E. Jaeckle.—p. 103.
Posterior Principal Plane of Optical System of Eye and Significance for Refraction. E. Tron.—p. 107.
Herpes Zoster Ophthalmicus: Report of Cases and Review of Literature. A. E. Edgerton.—p. 114.

Gonorrheal Syndrome without Gonorrhea.—The triad of urethritis, conjunctivitis and arthritis, occurring in this order, in the absence of a gonococcic etiology is known as Reiter's disease. The syndrome parallels gonorrheal infection so closely that suspicion of the gonococcus is difficult to dispel. In the case reported by Lucas and Weiss a crop of small vesicles developed chiefly in the central area of the cornea five days after the onset of the conjunctivitis. These broke down shortly to form discrete superficial ulcers which showed no tendency to coalesce or penetrate and healed without residual opacification within the ensuing three weeks. In this case sulfathiazole was given without a smear having been taken. Penicillin was administered repeatedly without benefit. Ultimate therapy consisted only of hot compresses and instillations of atropine. The prognosis for the eyes is good. In the reported case all symptoms had disappeared two months after hospitalization.

Bulletin of Los Angeles Neurological Society

10:1-84 (March-June) 1945

Alterations in Myelin Sheaths Adjacent to Traumatic Lesions of Brain. C. W. Rand and C. B. Courville.—p. 1.
Notes on Pathology of Cranial Tumors: I. Osteomas of Skull with Incidental Mention of Their Occurrence in Ancient Incas. K. H. Abbott and C. B. Courville.—p. 19.
Contribution to Study of Disseminated Encephalomyelitis: Report of 4 Cases Presenting Manifestations of Both Neuromyelitis Optica and Albuminocytologic Dissociation, One with Necropsy Findings. C. Marsh and S. S. DeVine.—p. 35.
Ancestry of Neuropathology: Monsieur Antoine Louis and His "Tumeurs Fongueuses de la Dure-Mère." C. B. Courville.—p. 46.

California and Western Medicine, San Francisco

63:55-106 (Aug.) 1945

Some Observations on Deficiency Disease, with Special Reference to Thiamine. J. F. Rinehart and L. D. Greenberg.—p. 63.
Lumbosacral Subarachnoid Block. C. E. Schuetz.—p. 64.
Public Health Bacteriology. J. C. Geiger.—p. 65.
Free Enterprise and the Doctor. P. A. Ferrier.—p. 67.
Role of Gallbladder in Disease of Liver. W. C. Boeck.—p. 69.
Infections of Liver, with Special Reference to Amebiasis. L. A. Alesen.—p. 73.
Portal Cirrhosis. A. M. Snell.—p. 74.
Status of Liver and Its Importance to the Surgeon. P. J. Cunnane.—p. 78.
Use of Products of Fibrinogen and Thrombin in Otolaryngology. H. P. Schenck.—p. 80.

Delaware State Medical Journal, Wilmington

17:129-146 (July) 1945

Practical Application of Rh Blood Type. J. W. Howard.—p. 129.
Skin Lesions Attending Use of Penicillin. L. B. Flinn, L. C. McGee, W. P. Featherston and D. O. Kern.—p. 133.
Periarteritis Nodosa. C. Levy.—p. 135.

Endocrinology, Springfield, Ill.

37:69-156 (Aug.) 1945

Tissue Aldehyde Shift in Rat Kidney. K. A. Oster, with technical assistance of Jean G. Baum.—p. 69.
Effects of Sex Steroids on Gonads of Starling. J. W. Burger.—p. 77.
Distribution of Cell Types in Anterior Hypophysis During Late Pregnancy and Lactation. N. B. Everett and B. L. Baker.—p. 83.
Prostate and Seminal Vesicle Response to Testosterone Propionate in Intact and Castrate Rats. Frances H. Steadman and B. Krichesky. —p. 89.
Cholesterol and Ascorbic Acid Content of Adrenal, Liver, Brain and Plasma Following Hemorrhage. G. Sayers, M. A. Sayers, Tsan-Ying Liang and C. N. H. Long.—p. 96.
Studies on Role of Liver in Metabolism of Progesterone. G. Masson and M. M. Hoffman.—p. 111.
Vicarious Metabolic Response: Influence of Blood from Thyroxinized Animals on Oxygen Consumption of Surviving Tissue. F. N. Craig and W. T. Salter.—p. 117.
Mitosis Stimulation in Thyroid Gland Induced by Thiouracil. K. E. Paschkis, A. Cantarow, A. E. Rakoff and M. S. Rothenberg.—p. 133.
Adrenal Medulla in Water Diuresis and Water Intoxication. R. Gaunt, Mildred Liling and Margaret Cordsen.—p. 136.
Studies on Water Intoxication in Adrenalectomized Rats and Influence of Desoxycorticosterone Acetate and Epinephrine in Water Diuresis. H. W. Hays and D. R. Mathieson.—p. 147.

Georgia Medical Association Journal, Atlanta

34:127-148 (July) 1945

Evolution of Medicine. C. Thompson.—p. 127.
Relief from Pain. W. A. Risteen.—p. 132.
Glaucoma: Its Diagnosis and Management. W. O. Martin Jr.—p. 135.

34:149-172 (Aug.) 1945

Neurosurgical Aspects of Lumbar and Sciatic Pain. E. F. Fincher. —p. 149.
Treatment of Morphine Addiction. J. C. Massee.—p. 154.

Illinois Medical Journal, Chicago

88:57-104 (Aug.) 1945

Hospital Care for Patient with Acute Poliomyelitis. Mary S. Sherman. —p. 71.
Mental Illness as Postwar Public Health Problem. C. S. Sommer.—p. 76.
Discussion of Lacrimal Problems. H. Gifford Jr.—p. 81.
Sulfathalidine: Clinical Evaluation in Infectious Diseases of Colon. M. H. Streicher.—p. 85.
Continuous Caudal Anesthesia. P. Pernworth.—p. 87.

Journal of Clinical Endocrinology, Springfield, Ill.

5:247-290 (July-Aug.) 1945

Pituitary Insulotropic Principle. J. W. Conn and L. Louis.—p. 247.
*Observations on 78 Thyrotoxic Patients Treated with Thiouracil. T. H. McGavack, A. J. Gerl, J. H. Morton, M. Vogel and D. Schwimmer.—p. 259.
Thiouracil in Control of Thyrotoxicosis. E. M. Watson.—p. 273.
Prolonged Administration of Diethylstilbestrol. K. J. Karnaky.—p. 279.

Thyrotoxic Patients Treated with Thiouracil.—McGavack and his associates report studies on 10 male and 68 female patients with thyrotoxicosis who were under treatment with thiouracil. Of these 78 patients 53 had a diffusely hyperplastic and 25 a nodular type of goiter. Periods of treatment varied from two weeks to sixteen months. "Nervousness," apprehension, palpitation and loss of weight were the most common symptoms and were among the first to disappear. Exophthalmos was present in 38 of the 78 patients and was decreased under treatment in 17 instances. In 65 instances the thyroid was initially enlarged. Under treatment there was a transient decrease in size in 20 and a more prolonged increase in size in 21. The auricular fibrillation initially present in 18 individuals disappeared in 12 without other therapy than the thiouracil. Eight of the 78 patients were continued on the drug only sufficiently long to prepare them for surgery. The basal metabolic rate was depressed by thiouracil in both the nodular and the hyperplastic type of thyrotoxicosis. The average initial basal metabolic rate for the entire group was plus 46 per cent, with a low of plus 18 and a high of plus 87. The approximate time taken for the basal metabolic rate to reach plus 15 or below was three weeks. In every instance the blood cholesterol rose as the basal metabolic rate fell. Toxic reactions included 2 cases of agranulocytosis and 2 cases with severe febrile reactions, chills and urticarial skin lesions. The maintenance dose of thiouracil has varied from 0.1 Gm. to 0.4 Gm. daily, with an average of 0.23 Gm. daily for the 67 patients whose initial basal metabolic rates were plus 30 or above. The treatment of 14 patients was successfully discontinued at the end of two and a half months in 5 cases, four months in 1, five months in 1, six months in 2, seven months in 2 and eight months in 3.

Journal of Immunology, Baltimore

51:1-64 (July) 1945

Inhibition of Phagocytosis by Penicillin. H. Welch, Ruth P. Davis and C. W. Price.—p. 1.
Serologic Reactions of Two Bacterial Levans. E. J. Hehre, Dorothy S. Genghof and J. M. Neill.—p. 5.
Administration of Certain Chemotherapeutic Agents to Mice by Incorporation of Chemical in Drinking Water: Evaluation of Method. S. D. Kramer and L. D. Bunch.—p. 15.
Studies on Relative Susceptibility of Young and of Full Grown Rabbits to Intravenous Infection with Hemolytic Streptococci. P. F. DeGara. —p. 23.
Use of Commercially Available Complement Fixing Antigen for Diagnosis of Elementary Body Types of Viral Infection. A. L. Florman. —p. 29.
Reduced Acute Toxicity of Antigens in Saline in Mineral Oil Emulsion. S. P. Halbert, J. Smolens and S. Mudd.—p. 39.
Studies on Hemolytic Streptococcus: VII. Comparison of Results of Immunization Against Scarlet Fever with Toxin and Aluminum-Adsorbed Toxoid of Streptococcus Scarlatina (NY5). M. L Menten, H. H. Finlay and Marie A. Andersch.—p. 45.
Immunologic Studies in Rheumatic Fever: I. Cutaneous Response to Type Specific Proteins of Hemolytic Streptococcus. B. Response to "Purified M" Proteins from Forty Known Types of Hemolytic Streptococcus Group A. L. M. Taran, J. M. Jablon and Helen N. Weyr. —p. 53.

Journal of Neurophysiology, Springfield, Ill.

8:221-274 (July) 1945

Effect of Anticonvulsant Drugs on Recovery of Function Following Cerebral Cortical Lesions. C. W. Watson and Margaret A. Kennard. —p. 221.
Chemical Changes in Cerebral Cortex Associated wtih Convulsive Activity. W. E. Stone, J. E. Webster and E. S. Gurdjian.—p. 233.
Functional Significance of Rostral Cingular Cortex as Revealed by Its Responses to Electrical Excitation. W. K. Smith.—p. 241.
Efferent Fibers of Parietal Lobe of Cat (Felis Domesticus). W. G. Gobbel Jr. and G. W. Liles.—p. 257.
Effects of Lowering Blood pH on Excitability of Nervous System. H. Koenig and R. A. Groat.—p. 267.

Journal of Urology, Baltimore

54:1-106 (July) 1945

Pathologic and Anomalous Conditions Associated with Duplication of Renal Pelvis and Ureter. R. Goyanna and L. F. Greene.—p. 1.
Bilateral Crossed Renal Ectopia: Case Report. C. M. Norfleet Jr. —p. 10.
*Renal Carbuncle: New Method of Treatment. T. P. Shearer, T. B. Wiper and J. M. Miller.—p. 12.
*Aneurysm of Renal Artery. B. Levine.—p. 17.
Enuresis in Young Male Adults. W. E. Forsythe Jr. and S. C. Karlan. —p. 22.
Bilharziasis of Bladder (Vesical Schistosomiasis). E. A. Ockuly.—p. 39.
Postoperative Progressive Gangrene of Skin Following Suprapubic Prostatectomy. S. H. Bassow.—p. 46.
Diverticulum of Female Urethra: Review of Literature with Case Report. I. G. Downer and F. D. Virgilio.—p. 53.
Filariasis in Armed Forces. R. A. Burhans.—p. 59.
Urologic Aspects of Filariasis. W. J. McMartin.—p. 62.
AP-43: New Antispasmodic for Use in Urology. C. L. Prince and E. J. Richardson Jr.—p. 75.
Experimental and Clinical Studies with Sulfacetimide (p-Aminobenzenesulfonylacetylimide) Its Toxicity and Efficiency in Bacillary Infections of Urinary Tract: I. Experimental Studies. D. Lehr. —p. 87.

New Treatment of Renal Carbuncle.—Shearer and his associates report that a man aged 20 had had pain of moderate severity in the left lower chest and upper abdomen for two months. The pain was associated with temperature of a septic type. The patient stated that four days before the onset of pain he had had several boils on his face and two carbuncles in the right cervical region. Gradually, pronounced tenderness became apparent in the left costovertebral region. Intravenous pyelography revealed some compression of the calix of the left kidney, which suggested a left perirenal abscess. At surgery an abscess was not present; further studies indicated an expansile mass in the lower portion of the kidney. At a second operation the kidney was freed to the hilar structures anteriorly and posteriorly, an aspirating needle was passed into several suggestive areas and purulent material was obtained from a site deep in the substance of the kidney at the junction of the upper two thirds and lower one third. Ten cc. of thick purulent material was released and a No. 16 F. catheter was sutured

into the cavity. Bacteriologic examination of this material established the presence of a hemolytic Staphylococcus aureus in pure culture. One Penrose drain was placed at the upper pole of the kidney and a second at the lower, the wound was closed in layers, 200,000 units of penicillin was administered intramuscularly daily, the abscess cavity was lavaged twice daily with 5 cc. of a solution of penicillin containing 250 units per cubic centimeter and rapid sterilization was effected.

Aneurysm of Renal Artery.—Levine observed a true aneurysm of the renal artery in a woman aged 59. The laminographic method of examination as first suggested by Wilhelm was particularly helpful in establishing the diagnosis. The aneurysm was approached transperitoneally. It was felt that in dealing with a friable aneurysm it might be well first to ligate the renal vessels before mobilizing the aneurysm and the kidney. As a precautionary measure, tapes were passed under the vena cava above and below the renal vessels. The transperitoneal approach is also to be preferred if repair of the aneurysm is contemplated. The patient has been examined at regular intervals for six months after the operation. During this time she has been free from all pain and has felt well.

Nebraska State Medical Journal, Lincoln

30:265-304 (Aug.) 1945

Types of Regressive versus Compensatory Mental Disorders. N. D. C. Lewis.—p. 268.
Education of Physicians: Now and After the War. V. Johnson. —p. 274.
American College of Surgeon's Program for Expansion of Graduate Training in Surgery. M. T. MacEachern.—p. 280.
American College of Physicians—Its Aims, Standards and Activities. E. R. Loveland.—p. 284.

30:305-340 (Sept.) 1945

Problem of Fractured Hip. G. W. Leadbetter.—p. 309.
Thiouracil in Treatment of Hyperthyroidism: Case Reports. G. W. Covey, F. L. Rogers and H. H. Whitlock.—p. 314.
Erythroblastosis Fetalis. C. F. Moon.—p. 318.
Psychosomatic Approach in Industrial Surgery. R. Thornell Mauer. —p. 320.
Rat Control. T. A. Filipi.—p. 322.

New England Journal of Medicine, Boston

233:287-314 (Sept. 6) 1945

Coarctation of Aorta: Experimental Studies Regarding Its Surgical Correction. R. E. Gross and C. A. Hufnagel.—p. 287.
Need of Facilities for Care of Feebleminded Infants and Young Children. I. V. Duguid.—p. 294.
Possible Interrelation of Psoriasis and Streptococcus Fecalis. J. H. Swartz.—p. 296.
Proctology. E. P. Hayden and T. A. Krolicki.—p. 297.
Epidural Gumma of Spinal Cord.—p. 305.
Epidermoid Carcinoma (Grade III) of Bronchus, with Metastasis to Regional Lymph Nodes.—p. 307.

New York State Journal of Medicine, New York

45:1711-1806 (Aug. 15) 1945

Treatment of Ménière's Syndrome. J. J. Rainey.—p. 1753.
Cardiac Dyspnea and Its Treatment. M. A. Donovan.—p. 1756.
Imperfections of Spinal Anesthesia. S. G. Hershey.—p. 1761.
Treatment of Acute Cholecystitis. G. W. Cottis.—p. 1765.
Radiography in Rib Fractures. R. W. Lewis.—p. 1767.
Instrument for Adult Circumcision. A. J. Philip.—p. 1768.

Northwest Medicine, Seattle

44:233-268 (Aug.) 1945

Etiologic Factors Responsible for T Wave Changes in Electrocardiogram. F. R. Maddison.—p. 237.
Cytologic Examination of Nasal Smears: Aid in Diagnosis of Chronic Nasal Sinus Disease. A. R. Miller.—p. 242.
Mandibular Fractures at King County Hospital Treated by Roger Anderson Skeletal Fixation. J. W. Graham.—p. 250.
Ingrowing Toenail. T. L. Hyde.—p. 252.
Control of Dysentery Outbreak in Vancouver Nursery School. S. P. Lehman and Ruth Jens.—p. 255.

44:269-302 (Sept.) 1945

Streptococcic Dissociation and Its Significance. E. E. Brown.—p. 272.
Tissue Reactions to Vitallium and Acrylic Implants. C. M. MacKenzie, D. H. Sharpless and P. Millard.—p. 277.
Plumbism. T. E. P. Gocher.—p. 283.
Cerebral Intoxication Result of Trichlorethylene. N. K. Rickles. —p. 286.
Inverse Relation of Nuclear and Cytoplasmic Function. W. B. Dublin. —p. 287.
Diagnostic Pointers in Blood Diseases. E. Podolsky.—p. 288.

Ohio State Medical Journal, Columbus

41:781-880 (Sept.) 1945

Brief Critique of Psychosomatics. C. H. Campbell.—p. 805.
Current Thinking in Nutrition. J. Forman.—p. 810.
*Treatment of Severe Acute Tonsillitis With and Without Sulfonamides. H. M. Clodfelter.—p. 819.
Neurosurgical Treatment of Gynecologic Disorders. E. W. Shannon. —p. 821.
Acute Necrotizing Angiitis Due to Hypersensitivity Following Sulfonamide Therapy. R. T. Thompson.—p. 824.

Treatment of Tonsillitis.—Clodfelter reports 400 cases of streptococcic sore throat selected from more than 3,000 acute upper respiratory infections of such severity as to require hospitalization. In 41 of the 175 treated with large doses of sulfonamides the course was similar to that of the untreated cases and they were discharged on the fifth or sixth day. The secondary rise in temperature was present in 134 of the sulfonamide treated cases. The greatest number of discharges from the hospital in this group was on the tenth day and the average number of hospital days was 9.7. Sulfathiazole and sulfadiazine were used in about an equal number of cases without any appreciable difference in the two drugs. The use of sulfonamides in the treatment of severe cases of acute tonsillitis results in a prompt drop in the temperature with some relief from symptoms. But there is usually a secondary rise in temperature at the time untreated cases are being discharged. The number of hospital days is increased from 5.3 to 9.7. The patients did not feel as well on the tenth day as the untreated ones did on the fifth. It seems highly suggestive that the use of sulfonamides in the treatment of acute streptococcic tonsillitis is not only without benefit but actually detrimental to the welfare of the patient.

Puerto Rico J. Pub. Health & Trop. Med., San Juan

20:419-548 (June) 1945

Antimalaria Measures for Protection of Military Personnel in Puerto Rico and Their Applicability to Civilian Malaria Control. J. M. Henderson.—p. 419.
*Sulfaguanidine in Treatment of Dysentery in Children. M. E. Wegman, A. Diaz Atiles, J. Basora Defilló, Elise G. Schlosser and Sophie D. Griffitts.—p. 473.
Schistosomiasis Mansoni Manifestations of Large Intestine. F. Hernández Morales.—p. 492.
Roentgenologic Changes of Small Intestine in Presence of Schistosoma Mansoni. F. Hernández Morales and G. Ruiz Cestero.—p. 507.
Reasons for Decline of Mortality from Puerperal Causes in Puerto Rico During Decade 1933-1943. J. S. Belaval.—p. 515.
Bacterias, Rickettsias and Pathogenic Virus Demonstrable with Electronic Microscope; Relations with Immunity and Chemotherapy; Morphology. S. Mudd and T. F. Anderson.—p. 529.

Sulfaguanidine in Dysentery.—Wegman and his associates studied the value of sulfaguanidine in young children. Immediately after admission a rectal swab culture was made and after this a culture was made of the first stool passed. Administration of sulfaguanidine was then begun with a daily dose of 0.1 Gm. per kilogram of body weight. Half of this daily dose was given on admission, followed by one fourth of the daily dose every six hours. The dosage was reduced to half the amount when diarrhea had been absent for two days and was discontinued entirely after four days without diarrhea. Forty of the children had had at least one culture positive for Shigella, Flexner V. W. Z or Sonne. Other varieties of Shigella were not encountered. Of the culturally positive cases, 23 were treated and 17 were untreated controls. The treated cases, regardless of the duration of the disease before treatment was begun, tended to terminate within a few days time, while a good proportion of the control cases lasted two weeks or more. In the 37 patients originally included in the study, but in whom cultures were never positive, there was little difference between the treated and the control cases. These observations are at variance with several reports in the literature which have indicated the value of sulfaguanidine even in the absence of positive cultures. The contrast in this study, however, adds to the evidence that sulfaguanidine must have a specific effect on Shigella. In the cases with positive culture, treatment with sulfaguanidine effected improvement both in terms of the duration of the diarrhea and in terms of the duration of the infection.

Rocky Mountain Medical Journal, Denver

42:561-634 (Aug.) 1945

Meaning of Medical School. A. Gregg.—p. 577.
Vascular and Neurologic Lesions Which Result from Exposure of Extremities to Moisture and Cold. L. J. Kleinsasser.—p. 580.
Surgical Treatment of Intertrochanteric Fractures. B. N. E. Cohn and V. R. Vonburg.—p. 587.
Treatment of Impetigo Contagiosa by Allantoin Sulfathiazole in Polyvinyl Alcohol Vehicle. F. V. Worman and E. S. Stong.—p. 592.
Colorado Division of Industrial Hygiene. A. T. Rossano Jr.—p. 595.

South Carolina Medical Assn. Journal, Florence

41:187-218 (Aug.) 1945

Toxemias of Late Pregnancy. J. D. Guess.—p. 187.
Bacillary Dysentery (Shigellosis). J. I. Waring.—p. 190.
Recent Progress in Medical Science. R. M. Pollitzer.—p. 192.
Bacteriologic and Other Studies in Public Aspects of Gonococcal Infection. H. Boatwright.—p. 196.

41:219-246 (Sept.) 1945

Relation Between Economic and Social Trends and Practice of Medicine. C. C. Carpenter.—p. 219.
Surgical Problems in Returning Veteran. E. B. Keck.—p. 222.
Proportion of Male to Female Live Births During Wartime. G. D. Johnson.—p. 225.
Intrathoracic Goiter. F. T. Wallace.—p. 226.

Texas State Journal of Medicine, Fort Worth

41:227-280 (Sept.) 1945

Anatomic and Mechanical Aspects of Treatment of Fractures of Forearm. W. G. Stuck.—p. 232.
Dietometabolic Urology. P. R. Stalnaker.—p. 241.
Virus Disease of Respiratory Tract. O. J. La Barge.—p. 244.
*Use of Aluminum Penicillin Mixtures in Maintenance of Blood Levels of Penicillin: I. Intramuscular Injections of Aluminum Penicillin. S. W. Bohls and E. B. M. Cook.—p. 249.
*Nebulized Penicillin in Treatment of Respiratory Infections. R. J. Hanks.—p. 253.
Use of Thiouracil in Treatment of Hyperthyroidism. E. E. Cooper.—p. 254.
Kerosene Poisoning. O. W. Lowery.—p. 259.
Venereal Disease Conference. G. P. Franks.—p. 261.
Vaginoperitoneal Fistula with Chronic Peritonitis Following Vaginal Hysterectomy. E. W. Bertner and K. C. Von Pohle.—p. 264.

Intramuscular Injection of Aluminum Penicillin.—Results obtained in treating patients with gonorrhea indicate that a single injection of 50,000 units or more of aluminum potassium sulfate and penicillin mixture will maintain a therapeutic level from six to nine hours. Two injections of 50,000 units at nine hour intervals would be the minimum dosage to maintain assayable levels for eighteen hours. In several cases of gonorrhea cures were obtained at low level dosage, but the number is far too few to justify conclusive statements. The practicability of the use of aluminum penicillin mixtures in decreasing both dosage of penicillin and number of injections is evident. Subsequent tests with aluminum penicillin and other therapeutic agents may reveal that syphilis as well as gonorrhea may be treated by general practicing physicians without hospitalizing the patient in rapid treatment centers.

Nebulized Penicillin in Respiratory Infections.—Results with nebulized penicillin have been so much superior to those with sulfonamides that there is no need to use the latter with the attendant danger of sensitization and possible death. Thus far no complications have been experienced with penicillin. A saline solution containing 500 units per cubic centimeter seems to give the best results. An oxygen tank with a flow meter and humidifier is connected by a rubber tube to a DeVilbiss Nebulizer No. 40. Two cc. of penicillin solution is placed in the glass nebulizer, and an oxygen flow of 4 liters is set. The patient is instructed to hold the nebulizer between the teeth and breathe through the mouth. The treatment usually takes twenty minutes and is given once a day. In acute tonsillitis, laryngitis and bronchitis the results are startling. One, two or three treatments gives complete relief from fever and other symptoms. The subacute type of infection, such as the streptococcic pneumonitis, will respond to six to twelve treatments given once a day. The chronic infection will require twenty to sixty treatments once daily. In cases of chronic infection the sputum will be reduced, the temperature will return to normal or nearly so, the toxic symptoms will diminish and a feeling of well being will be reestablished. Virus pneumonia will also respond to this method of treatment even where parenteral injection of penicillin has failed.

FOREIGN

An asterisk (*) before a title indicates that the article is abstracted below. Single case reports and trials of new drugs are usually omitted.

British Medical Journal, London

2:205-242 (Aug. 18) 1945

*Trial of Whooping Cough Vaccine in City and Residential Nursery Groups: Report to Medical Research Council. A. M. McFarlan, Elizabeth Topley and Mary Fisher.—p. 205.
Problems of Naval Warfare Under Climatic Extremes. M. Critchley.—p. 208.
Preservation of Human Spermatozoa at Low Temperatures. A. S. Parkes.—p. 212.
Hemolytic Streptococcal Throat Infections: Their Complications and Sequels, with Special Reference to Penicillin Treatment. P. Robinson.—p. 213.
Two Unusual Stress Fractures. I. D. Kitchin and D. A. Richmond.—p. 214.
Tuberculin Testing in Children: Comparison of Methods. D. Bell and Ursula Jerram.—p. 215.
Postoperative Vomiting in Relation to Anesthetic Time. J. M. Smith.—p. 217.

2:243-272 (Aug. 25) 1945

Sequelae of Cerebrospinal Meningitis. Follow-Up Study of 986 Cases. J. A. Degen, with assistance of Helen Cameron, Virginia L. M. Robinson and Mildred S. R. Wieden.—p. 243.
Review of Psychologic Work at Brain Injuries Unit, Edinburgh, 1941-1945. O. L. Zangwill.—p. 248.
Accidental Infant Suffocation. W. H. Davison.—p. 251.
*Polyneuritis After Jungle Sores: Series of 21 Cases. R. L. Ward and A. S. Mason.—p. 252.
Acute Infective Polyneuritis. C. P. Petch.—p. 254.
Premedical Zoology. M. Abercrombie and M. L. Johnson.—p. 262.

Whooping Cough Vaccine in Nursery Groups.—McFarlan and his associates assessed the prophylactic value of pertussis vaccine by comparing inoculated children and control children. Over 600 susceptible children of 6 months to 3 years of age attending welfare clinics and wartime day nurseries in Oxford City, and 110 evacuee children of 6 months to 3 or 4 years of age distributed in eight residential nurseries in Oxfordshire, Berkshire and Buckinghamshire were divided into "inoculated" and "control" groups by methods designed to render the two groups similar in every respect other than inoculation. The inoculated children were given either two doses of pertussis vaccine at a four weeks interval or four doses at intervals of one, one and four weeks. The total dosage ranged from 40,000 to 100,000 million organisms, corresponding to an American dosage of about 120,000 to 300,000 millions. Whooping cough occurred sporadically in Oxford City during the succeeding eighteen months and broke out in four of the residential nurseries two weeks to nine months after the last inoculation. Significant differences were not observed in the incidence or severity of whooping cough between the inoculated and the control children. In Oxford City 12.5 per cent of 327 inoculated and 14.1 per cent of 305 control children developed definite whooping cough. In the residential nurseries the corresponding figures were 55 per cent of 33 inoculated and 63 per cent of 30 control children. The evidence obtained from this investigation lends no support to the view that pertussis vaccine is of value in the prophylaxis of whooping cough; and it is suggested that the use of this vaccine should be discontinued till some positive evidence of its value is obtained.

Polyneuritis After Jungle Sores.—Ward and Mason say that in a recent campaign in the Burmese jungle many of the British and Indian troops developed multiple indolent ulcers, commonly known as "jungle sores." Some of those who had suffered from jungle sores contracted a peripheral neuritis later. Of 21 patients 11 were admitted complaining of symptoms referable to their neuritis, 9 because of malarial relapse and 1 with jungle sores. All had suffered from jungle sores but, except for patient 19, the ulcers were healed at the time of admission. Sixteen patients reported blurring of vision as the first symptom of nervous involvement. The average duration of blurred vision was four weeks. All patients noted disturbance of sensation. In 4 cases it synchronized with blurring of vision, but in the remainder it occurred three weeks later. Sensory symptoms consisting of tingling, numbness and coldness of the extremities were often severe enough to make the men report sick. In the milder cases these paresthesias were the only complaints; however, others later complained of difficulty

in walking and writing. The picture in its most severe form was completed by progressive weakness of the limbs, spreading to involve hips and shoulders. In addition anesthesia of the extremities, astereognosis and ataxia were present. From a shuffling walk with feet apart the gait deteriorated until walking was impossible. Examination of the nervous system in these cases showed the characteristic signs of a polyneuritis. Eleven of the cases were mild. In 7 moderately severe cases the disability was sufficient to impede the patient's everyday life. Only 3 patients became bedridden. The polyneuritis was not influenced by the vitamin B complex. Moreover, the nutrition of all patients was good. The author suggests that a toxin, elaborated by bacteria infecting the ulcers, was the responsible agent. He thinks it most likely that the polyneuritis followed a diphtheritic infection of the jungle sores.

Indian Journal of Medical Research, Calcutta

32:109-252 (Oct.) 1944. **Partial Index**

Vitamin B_2 Deficiency as Cause of Eye Diseases in Bengal. E. O. Kirwan, K. Sen and N. Bose.—p. 109.
Estimation of Pyridoxine (Vitamin B_6) in Foods Using Rice-Moth Larvae (Corcyra Cephalonica St.). P. S. Sarma.—p. 117.
Antithiamine Factor in Carp. K. Bhagvat and P. Devi.—p. 123.
Inactivation of Thiamine by Certain Foodstuffs and Oil Seeds. K. Bhagvat and P. Devi.—p. 131.
Liberation of Tyrosine, Tryptophan, Cystine and Arginine from Proteins. K. Bhagvat and V. V. Sreeramamurthy.—p. 145.
Egg-White Injury (Induced Biotin Deficiency) in Rice-Moth Larvae (Corcyra Cephalonica St.). P. S. Sarma.—p. 149.
Studies on Destruction of Vitamin A in Shark Liver Oil. S. D. Rao. —p. 155.
Vitamin C in Pine Needles. N. K. Iyengar. B. C. Bose and B. Mukerji.—p. 165
Method for Demonstration of Negri Bodies. S. McDonald.—p. 205.
*Protozoan Parasite of Central Nervous System. N. Veeraraghavan. —p. 207.
Isolation of Strain of Rickettsia Orientalis from Cases of XK Typhus in Ceylon. R. B. Lucas.—p. 223.
In Vitro Test for Assay of Potency of Cobra Antivenene. M. L. Ahuja and A. G. Brooks.—p. 227.
Studies on Experimental Fluorine Poisoning in Rats. S. Ranganathan. —p. 233.
Studies in Vital Capacity. D. V. S. Reddy and P. B. Sastry.—p. 237.

Protozoan Parasite of Nervous System.—Veeraraghavan encountered forms in the midbrain of guinea pigs experimentally infected with rabies which have not, apparently, been previously described. The peculiar forms have been investigated over the past six years. The conclusion was reached that they are due to the presence of a parasite, probably protozoal in character, which is specific to the central nervous system. It has not been possible to identify this parasite with any previously described, such as Encephalitozoon cuniculi, or any known species of toxoplasma. The protozoal parasite was first encountered in the midbrains of guinea pigs infected with the jackal strain of rabies virus. The parasite, with the exception of certain cystic forms of doubtful significance, invariably occurred in the cytoplasm of nerve cells, particularly those ventral to the central canal of the midbrain. In a few cases, parasites were found also in the Purkinje cells of the cerebellum and in the hippocampus major. The smallest forms of the parasite encountered were minute "spores." They were regularly found in the midbrains of the naturally infected dogs and jackals and in the brains of guinea pigs inoculated with suspensions of their brains. The next stage of the parasite appeared to be the "ring" form. It might be round or oval, and it was variable in its position within the vacuole. Certain forms resembling the accolé forms of the malaria parasite were occasionally found. These consisted of a crescentic bridge of cytoplasm with a rounded chromatin dot. In slightly older forms the chromatin mass was slightly larger, the protoplasm was thickened and the parasite assumed a variety of shapes. Segmentation of the chromatin appeared to be the next stage in the development of the parasite. In the segmenting forms the protoplasm appeared to be a solid mass with a varying number of chromatin dots. No evidence was found to suggest that the various forms of the parasite were the result of alterations in the constituents of nerve cells. The possibility of the parasite being a natural infection common to dogs and jackals, unconnected with rabies infection, was investigated. Neither parasites nor Negri bodies were ever encountered in animals without rabies. Certain stages in the life cycle of the protozoal parasite described appear to be "filtrable"; it has been possible to produce evidence of infection in experimental animals by the inoculation of brain suspensions passed through Berkefeld V and N candles. The protozoal parasite appears to be a specific infection of the central nervous system, and in spite of exhaustive searches it has never been observed in other organs and tissues. It was not observed except in association with natural or experimental rabies infection. The suggestion has been put forward that Negri bodies may represent a stage in the life cycle of the parasite.

Lancet, London

2:129-160 (Aug. 4) 1945

Return of Doctor: Address to British Medical Association. H. S. Souttar.—p. 129.
Epidemiology of Acute Poliomyelitis in India Command. D. McAlpine. —p. 130.
Assessment of Male Fertility by Semen Analysis: Attempt to Standardize Methods. Clare Harvey and Margaret Hadley.—p. 134.
Glomangioma: Report of 3 Cases. W. M. Beattie.—p. 137.
Secondarily Infected Liver Abscess Treated with Penicillin. R. S. Hunt. —p. 138.
Case of Syphilitic Wrist Drop. N. Hobhouse.—p. 141.

2:161-192 (Aug. 11) 1945

Effort Syndrome. I. G. W. Hill and H. A. Dewar.—p. 161.
Costoclavicular Compression of Brachial Plexus and Subclavian Vessels. A. D. LeVay.—p. 164.
Natural Safeguards in General Anesthesia. W. B. Primrose.—p. 166.
Vaccinia Virus: Immunologic Unity of Different Strains. E. S. Horgan and M. A. Haseeb.—p. 170.
*Function of Autolytic Enzymes in Bacteriolysis by Penicillin. E. W. Todd.—p. 172.
Hematuria Associated with Hemorrhagic Diathesis. R. W. Evans and H. C. MacLaren.—p. 175.

Autolytic Enzymes in Bacteriolysis by Penicillin.—Todd shows that autolysis plays a large part in the lysis which occurs when organisms are killed by penicillin; they do not however entirely exclude the possibility that penicillin may have bacteriolytic properties apart from autolysis. The rate of lysis of bacteria in cultures containing penicillin depends on the production of autolysin. Organisms which produce large quantities of autolysin are lysed rapidly; those which produce less autolysin are lysed more slowly, while those which produce no autolysin are probably not lysed at all. Living bacteria are resistant to autolysis; they become sensitive when killed by penicillin, by heat or by suspension in isotonic solution of sodium chloride. The most rapid bactericidal action of penicillin occurs when organisms are at their maximal rate of multiplication. The rate of bacteriolysis in cultures containing penicillin is therefore governed by both the rate of multiplication and the production of autolysin. Certain bacteria which produce little autolysin are killed by penicillin without undergoing bacteriolysis; they are then rapidly lysed if autolysin is added to the dead culture. The most probable explanation of bacteriolysis in cultures containing penicillin is that the organisms are first killed by penicillin and then autolysed.

2:193-224 (Aug. 18) 1945

Neurosurgery in Eastern Theater of War. R. T. Johnson and R. C. S. Dick.—p. 193.
Nicotinamide Methochloride Elimination Tests on Normal and Nicotinamide Deficient Persons. P. Ellinger, R. Benesch, with clinical addendum by S. W. Hardwick.—p. 197.
Polyarteritis Nodosa Developing During Antisyphilitic Treatment. H. G. Miller and M. G. Nelson.—p. 200.
*Intestinal Mucosa in Ulcerative Colitis. A. M. Gill.—p. 202.
Psychiatric Factors in Peripheral Vasoneuropathy After Chilling. J. W. Osborne and J. Cowen.—p. 204.
Selection of Patients for Blood Transfusion. H. Conway.—p. 206.

2:225-260 (Aug. 25) 1945

Teaching of Medicine. R. S. Aitken.—p. 225.
Education of a Surgeon. W. H. Ogilvie.—p. 229.
Training of General Practitioner. H. Crichton-Miller.—p. 231.
Visual Education. A. S. V. Thomas.—p. 234.
Health Education Through Isotype. O. Neurath.—p. 236.

Intestinal Mucosa in Ulcerative Colitis.—The course of ulcerative colitis with its tendency to spontaneous remissions and relapses led Gill to consider that in some cases at least the condition might be a deficiency disease. Preliminary inves-

tigations suggested that the missing hypothetical factor might be present in or produced by the intestine. Feeding experiments in 1 case over several years showed that remissions could be induced regularly by giving uncooked pig's small intestine by mouth. Relapses always followed when this treatment was stopped. Remissions have also been induced in other cases in which similar raw material was given; others, however, have shown no response. Pig's small intestinal mucosa, dried and defatted, given by mouth in powder form, has also induced remissions in some cases. Those patients who respond relapse when the treatment is stopped. Of the dried mucosal preparations tried, one has little or no effect, whereas the other appears to be as effective as the raw material. Experiments with a parenteral extract have so far been unsuccessful. The results obtained with this treatment do not appear to be coincidental or psychologic: they are compatible with the deficiency theory advanced, but a local effect in the colon is not yet excluded. The author thinks that the term "ulcerative colitis" must cover more than one disease, or that more than one etiologic factor is responsible for the syndrome, since it is impossible to forecast whether or not a given case will respond to this form of treatment.

Acta Medica Scandinavica, Stockholm

120:1-193 (Jan. 15) 1945. **Partial Index**

Preexcitation and Auricular Fibrillation. S. Åkesson.—p. 1.
*Prognosis of Essential Hypertension, with Remarks on Indications for Operative Treatment. H. Rasmussen and J. Bøe.—p. 12.
Importance of Duodenal Glands of Brunner to Formation of Red Blood Corpuscles in Swine. C. L. S. Bohn, E. Landboe-Christensen and C. M. Plum.—p. 32.
Distribution of Poliomyelitis Virus in Intestines of Normal Mice. S. Gard.—p. 40.
*Cardiac and Respiratory Neurosis After Contusions of Chest Wall. H. Heckscher.—p. 53.
Role of Tyrosine in Pernicious Anemia. E. Jacobsen and C. M. Plum. —p. 81.
After-Examination of Surgical and Nonsurgical Cases Showing "Clinical Symptoms of Herniated Disk." L. Kirstein.—p. 93.
Rhythmic Variations of Liver Glycogen and Pyruvic Acid of Blood in Experimental Obstructive Jaundice. Y. Edlund and H. Holmgren. —p. 107.
*Gastritis. A. Holtermann and H. Myhre.—p. 130.

Prognosis of Essential Hypertension.—One hundred patients with hypertension studied in 1936 to 1938 were reexamined in 1943 with consideration of the prognostic value of the earlier pathologic findings in the cardiovascular and renal systems. Fifty-two patients had died, 44 were still living and 4 had not been traced. Of the 44 survivors, 36 were women and 8 were men. The cause of death of 2 patients was unknown, while 13 died of cardiac insufficiency (26 per cent of the number of deaths), 9 died of myocardial infarction (18 per cent of the deaths), 19 (38 per cent) died of apoplexy and 3 (6 per cent) died of uremia. While the prognosis of myocardial infarction and uremia may be difficult, as they show no distinct relation to the earlier findings in the cardiovascular and renal systems, cardiac insufficiency exhibits a distinct correlation to the size of the heart and to the electrocardiographic alterations (left ventricular hypertrophy). The apoplexies show especially clear relationship to the height of the blood pressure, both the systolic and particularly the diastolic. Nearly one third of those with systolic pressure above 200 and more than one third of those with diastolic pressure above 125 died of apoplexy during the observation period. The surgical treatment of hypertension may in these groups be considered from the standpoint of prophylaxis of cerebral hemorrhage. Reinvestigation of the blood pressure showed that it was increased in only 5 patients. Most often it is stationary and in one fourth of the patients distinctly reduced, while in 2 patients it has become normal. Although in 1 patient there has developed nephrosclerosis, the 39 patients reexamined show primarily a slow development of hypertensive heart disease, sometimes with myocardial infarction and angina pectoris, sometimes with cardiac enlargement, this disease being electrocardiographically characterized by development of left ventricular hypertrophy in different stages.

Traumatic Cardiac and Respiratory Neurosis.—Heckscher presents a survey of a series of 22 patients suffering from the sequelae of thoracic contusions. Twenty of these patients were men and 2 were women. They were betweeen the ages of 33 and 60. Most of the complaints were not caused by any organic heart lesions but by the bruises inflicted on the chest wall and by a traumatic cardiac and respiratory neurosis. The symptoms of this traumatic neurosis were very much like the symptoms of the nontraumatic cardiac and respiratory neurosis. Conspicuous subjective symptoms were dyspnea on exertion or paroxysmal shortness of breath at rest, a feeling of oppression, palpitations, pain in the chest or in the back, anxiety and in some cases pronounced hypochondriasis or a real anguish. The objective symptoms were those which together form the pathognomonic triad of pulmonary emphysema: (*a*) the respiration changed from abdominal to mixed or purely thoracic type, (*b*) the changed posture, i. e., postural anomalies and (*c*) widening of the thoracic cavity and expansion of the lungs. In addition some patients presented the special neurotic type of respiration with sporadic, forced inspirations followed by delayed return to normal level. Some patients presented a mutual interdependence of pulse rate and respiration, both being influenced decidedly more by psychic alterations than by physical exercise, so that paradoxical reactions to tests of heart function could be seen, quite unlike the reactions of patients suffering from organic heart failure. The pains in the chest or back presented by the majority of the patients were partly caused by the bruised muscles of the chest wall and the back and partly by the functional changes in the muscles overstrained by static work. As these patients had displayed habitual anomalies of posture even before the accident, it is likely that they have contributed to the development of the disease. Differential diagnosis between functional and organic disorders of the heart must be based on the absence or the presence of symptoms absolutely pathognomonic for an organic heart disease. An erroneous diagnosis of organic heart failure may lead to a lasting invalidism of the patient. Physical therapy with exercises instituted soon after the accident may be of considerable help in preventing fixation of the chest, eliminating at the same time the tendency to persisting functional disturbances.

Gastritis.—Holtermann and Myhre found that gastritis accompanied duodenal ulcer in every case. In nearly half of their ulcer cases the gastritis was of hypertrophic type. In certain cases gastritis may consist only of a reddening and mild edema caused by hyperfunction. After a dietary course ulcer patients with hypertrophy and superficial catarrh may be improved with regard to the superficial catarrh, but hypertrophy will persist in the form of thickening and irregularity of the folds. Gastritis associated with duodenal ulcer was generally more pronounced and diffuse than that in gastric ulcer in which the mucosa may present a normal aspect after a successful dietary course. The subjective symptomatology of gastritis varies considerably. Sedimentation tests were performed in 135 cases. These tests revealed that an increased amount of mucous sediment formed within twenty-four hours in the gastric juice of fasting patients is to be considered as an important objective symptom. A series of nitrogen determinations in the gastric juice from fasting persons showed little correlation with the gastroscopic and roentgenologic findings and also with the results of the sedimentation tests. Nitrogen determination is of little value as a diagnostic sign. Gastroscopy is the most reliable diagnostic method and may likewise aid in classification. The gastritis may also be demonstrated roentgenologically. Better information will be obtained when small amounts of contrast medium are spread evenly over the mucosa. The method used by the authors consists in aspirating the gastric juice of the fasting patient with a duodenal tube during fluoroscopy. The mucosal folds must not be separated by outside pressure, and the supine position of the patient is preferable to the prone position. An irregular course of the folds often suggests gastritis, especially if it is associated with a certain degree of thickening. To evaluate the thickening of the folds, the form and size of the stomach are to be considered likewise. There is a fair chance of a correct diagnosis with an ordinary angular stomach. Negative roentgenologic findings do not exclude gastritis. Differential diagnosis between hypertrophic gastritis and superficial catarrh and atrophy should not be based on roentgenologic findings.

Book Notices

A Manual of Surgical Anatomy. Prepared Under the Auspices of the Committee on Surgery of the Division of Medical Sciences of the National Research Council. By Tom Jones and W. C. Shepard. Cloth. Price, $5. Pp. 195, with illustrations. Philadelphia & London: W. B. Saunders Company, 1945.

This is the last volume of the Military Surgical Manuals edited by the Committee on Surgery of the National Research Council for medical officers of the armed forces. While victory has fortunately diminished the opportunities for its use as a field manual, it will have a much more lasting value than its predecessors in the series. It was originally planned as a concise atlas of applied surgical anatomy of a format that would make it easy to carry and use under campaign and battle conditions. It is a visual presentation of anatomic regions most likely to be of importance to the surgeon caring for lesions produced by weapons of warfare. The material for presentation has been well selected from that point of view. The illustrations are new, having been redrawn from older cuts or evolved specially for this manual. They are line drawings, in many of which color has been skilfully used to aid greatly in the visual effects. The drawings are large enough to be easily studied, while the labels in English terminology are printed in large type so as to be clearly readable. The contents are arranged in four sections: part I, the head and neck; part II, the trunk-thorax, abdomen and pelvis; part III, the upper extremity; part IV, the lower extremity. Surgical approaches presented with each of these sections show with accuracy and clarity the approved avenues of approach used in the surgery of today. The incisions shown for operations on the thorax are especially valuable, as they were developed for this book and brought together for the first time in these illustrations. It is hoped that in the next edition the abdominal wall will receive similar treatment. There is an explanatory index in which are listed and defined the anatomic structures shown in the charts; the Standard English and B. N. A. terms are both given when there is a difference of anatomic name. That the book is the result of the collaboration of Mr. Tom Jones and Mr. W. C. Shepard in a field of surgical illustrations assures immediately a work of a high quality, and they are to be congratulated on the result of their labor, carried out with great zeal and haste in the face of national emergency. While the urgency for the manual has disappeared, the need for such a manual in civilian surgery remains. Anatomy cannot be learned from books, but such an atlas serves always as a convenient refresher to the surgical mind, and the surgical approaches are a matter of interest to all surgeons. This manual is heartily recommended to students of surgical anatomy, to those studying surgery and to those practicing the surgical arts.

The Principal Nervous Pathways: Neurological Charts and Schemas with Explanatory Notes. By Andrew Theodore Rasmussen, Ph.D., Professor of Neurology, Department of Anatomy, University of Minnesota Medical School, Minneapolis, Minn. Third edition. Cloth. Price, $3.50. Pp. 73, with 28 illustrations. New York: Macmillan Company, 1945.

This excellent monograph of nervous pathways should be in the possession of every neurologist, neurophysiologist and neurosurgeon. Former editions have proved on many occasions to be most helpful in cerebrospinal localization because the author has followed the concept of correlation of function and structure in the central nervous system, which should be the method of teaching. The illustrations speak for themselves. The work is the best of its kind for ready reference and study and is highly recommended.

A Manual of Ophthalmology for Medical Officers. By Lt.-Col. B. W. Rycroft, M.D., D.O.M.S., F.R.C.S., Surgeon and Dean, Royal Eye Hospital, London. Cloth. Price, $2.50. Pp. 95, with 59 illustrations. New York & London: Paul B. Hoeber, Inc., 1945.

This book is an outline presenting in brief form the methods of examination and the common diseases of the eyes and eye injuries seen in military practice by the general medical officer. It is of little value to the practicing ophthalmologist. It might be useful to the industrial surgeon who does not have a consulting ophthalmologist available.

Mental Disorders in Later Life. Edited by Oscar J. Kaplan. Cloth. Price, $5. Pp. 436, with 14 illustrations. Stanford University: Stanford University Press; London: Oxford University Press, 1945.

The increasing number of books dealing with the problems of old age bears testimony to the growing interest in the subject. Dr. Kaplan well describes the nature of this book in the preface when he says that it does not pretend to be an exhaustive treatment of the subject of mental disorders in later life but "is rather a collection of essays selected to indicate the level of present achievement in this area. . . In this book we see the pattern of future attack upon the mental diseases of later life. . . through the close collaboration of psychiatrists, statisticians, physiologists, sociologists and psychologists." Seventeen contributors have written separate chapters, which cover virtually all phases of the subject. Of particular interest are the chapters on the physiologic and the psychologic aspects of aging, on the neuroses of later maturity, on the senile psychoses and cerebral arteriosclerosis, on psychotherapy in the older person and on mental hygiene in later maturity. This book sounds a more optimistic note for the aging and aged patient than most publications in this field. It emphasizes the point that much more can be done for the aging than for the aged and that the time to prepare for old age is while one is still in the vigor of maturity. The book is a mine of information for the physician who wants to learn more about recent important advances that have been made in understanding the senescent and the senile patient.

Klinische und erbbiologische Untersuchungen über die Heredoataxien. Von Torsten Sjögren. Acta psychiatrica et neurologica Supplementum XXVII. Paper. Price, 15 Dän. Kron. Pp. 200, with 6 illustrations. Copenhagen: Ejnar Munksgaard, 1943.

This is a monograph on the hereditary ataxias written by the author from material studied in Göteborg and Stockholm. There are six chapters: the material and its classification, the collection of the author's cases, clinical analyses, hereditary studies, summary and bibliography. This is an excellent contribution in relation to genealogical and clinical studies in 188 cases. Three thousand persons were studied from the hereditary point of view.

The Chemical Formulary: A Collection of Valuable, Timely, Practical Commercial Formulae and Recipes for Making Thousands of Products in Many Fields of Industry. Volume VII. Editor-in-Chief: H. Bennett. Cloth. Price, $6. Pp. 474. Brooklyn: Chemical Publishing Co., Inc., 1945.

As stated in the preface, the Chemical Formulary is a collection of valuable and practical formulas and recipes, compiled with the assistance of many contributors from numerous fields of industry. As in other volumes the methods of preparation are given in many instances, which is of considerable aid. The book is divided into many parts relative to the different types of formulas. This volume, with its various new data and the enlargement of the directory of sources of chemicals and supplies, will prove to be of great value in the technical field. However, the book lends little value to the physician but a hand to medical quackery. It is to be commended in that it makes no claims for remedial action and thereby assumes no responsibility for its therapeutic uses.

Diseases of the Nervous System Described for Practitioners and Students. By F. M. R. Walshe, O.B.E., M.D., D.Sc., Physician in Charge of the Neurological Department, University College Hospital, London. Fourth edition. Cloth. Price, $4.50. Pp. 360, with 51 illustrations. Baltimore: William Wood & Company, 1945.

This tremendously practical and easily understandable book should become more popular in the medical schools of the United States. It is made up of twenty-nine chapters divided into two parts. Part one takes up the general principles of neurologic diagnosis and part two gives a descriptive account of the more common diseases of the nervous system. The book is written for use by the student and the general practitioner. It should be in the office of all medical men. The author has written his book from a practical standpoint so that the reader will not have to remember individual symptoms of each nervous disease but has to learn those symptoms which occur in lesions of various structures or systems. This book is highly recommended.

Queries and Minor Notes

THE ANSWERS HERE PUBLISHED HAVE BEEN PREPARED BY COMPETENT AUTHORITIES. THEY DO NOT, HOWEVER, REPRESENT THE OPINIONS OF ANY OFFICIAL BODIES UNLESS SPECIFICALLY STATED IN THE REPLY. ANONYMOUS COMMUNICATIONS AND QUERIES ON POSTAL CARDS WILL NOT BE NOTICED. EVERY LETTER MUST CONTAIN THE WRITER'S NAME AND ADDRESS, BUT THESE WILL BE OMITTED ON REQUEST.

THE THIRD STAGE OF LABOR

To the Editor:—**Is there really a third stage of labor or is it simply a continuation of the second stage? Is it not a fact that with the final contraction which expels the fetus the placenta is completely severed from the uterine wall and then acts only as a foreign body in the uterus and should be manually expelled at once?**

P. V. Janse, M.D., Algona, Iowa.

ANSWER.—The third stage of labor comprises two phases, separation and expulsion. The normal mechanism rarely separates the placenta completely as the baby leaves the uterus. In many instances this undoubtedly happens, so that the phase of separation can be designated as part of the second stage. More often partial separation takes place at the time of expulsion of the fetus. Complete separation is accomplished by resumption of uterine activity. The expression of the placenta should not take place until evidences of complete separation are apparent. Premature attempts at the expression of an incompletely separated placenta may lead to uterine atony and excessive bleeding. Mismanagement of this phase of the third stage is the most common cause of postpartum hemorrhage.

The ineffective third stage of labor has led to the use of oxytocic drugs during this period. To accomplish complete separation of the placenta at the time the baby leaves the uterus, M. Edward Davis (The Use of Ergonovine in the Placental Stage of Labor, *Am. J. Obst. & Gynec.* **43**:775 [May] 1942) has suggested the administration of 0.2 mg. of ergonovine intravenously just as the head is being delivered. Twenty or thirty seconds should intervene before the rest of the baby is born. The powerful uterine contraction initiated by the ergonovine results in the immediate and complete separation of the placenta, as the placental site shrinks because of the uterine contraction. Expression of the placenta should follow immediately. Improper timing of this procedure may result in the incarceration of the placenta in a contraction zone at the lower portion of the uterus.

This artificial conduct of the third stage has had extensive clinical experience during the last five years. It has resulted in a great decrease in the blood loss during placental separation, as well as in a definite reduction in the complications associated with the placental stage.

CESSATION OF MIGRAINE DURING PREGNANCY

To the Editor:—**A woman who has been subject to migraine became pregnant for the first time after nineteen years of married life and delivered a normal baby. The baby is now 3 weeks old and the headaches, after absence during the pregnancy, have returned.**

J. Huntley Boyd, M.D., Trenton, Mich.

ANSWER.—Among women with migraine it is the rule for headaches to occur in relation to menstruation, to be absent during pregnancy and to fade out at the menopause. These circumstances point to the importance of the sex hormones in causing migraine attacks and suggest that appropriate endocrine therapy or procedures which would neutralize the effect of the menstrual cycle on the sympathetic nervous system would be of value in the treatment of these patients. Unfortunately there do not seem to be any consistently encouraging reports of endocrine therapy. Clinical research on this subject is long overdue.

EDIBLE FROGS

To the Editor:—**We have in our locality two species of frog: One is the green head and is highly esteemed as food; the other is the leopard frog, which has no green coloring. As youngsters we referred to the latter variety as the "stink" frog. The habits of the two varieties so far as I know appear to be identical. Is there any reason why the leopard variety would not be a safe article of food?**

W. C. Hensyl, M.D., Berwick, Pa.

ANSWER.—The leopard frog is eaten in several parts of this country. There seems to be no reason for its being less highly esteemed than the green frog. The other large species of the genus Rana are also edible.

Reference:

Wright, A. H.: Frogs: Their Natural History and Utilization, U. S. Bureau of Fisheries, Doc. 888, 1920, pp. 1-44.

MECHANISM OF DIABETIC COMA

To the Editor:—**It has been stated that a lack of insulin is the fundamental cause of diabetic coma. The lack of insulin causes a reduced glucose oxidation throughout the body. This reduced glucose oxidation is particularly manifest in those organs which burn glucose practically exclusively for energy purposes such as the brain. For this reason it would be proper to state that the immediate cause of diabetic stupor and coma is a reduced glucose oxidation by the brain. A similar mechanism is present in hypoglycemic coma, namely a reduced glucose oxidation resulting from the reduced delivery of glucose to the brain. The final common effect on the brain will be the same, therefore, in diabetic and hypoglycemic coma, and the common factor is a pathologically reduced glucose oxidation. Diabetic coma may not be attributed to ketone bodies, because these are not always present in pathologic amounts. Further, they may be present in pathologic amounts in diabetes without coma. The ketosis is secondary to the deranged carbohydrate metabolism and disappears when the deranged metabolism is restored with insulin. Ketosis may also be present in hypoglycemic states because of the reduced glucose oxidation. Reduced glucose utilization is always accompanied by reduced oxygen consumption. This has been shown by several investigators on both man and the dog as well as on isolated tissues. Cells consume oxygen in proportion as they utilize glucose. Restoration of glucose oxidation in diabetic coma by insulin is apparently brought about by glycogen synthesis in the liver. The liver in diabetic coma has a low glycogen content. With insulin the glycogen stores are built up. It is believed by some that such glycogen breaks down, yielding gamma glucose, a form which is readily oxidized by the brain and other tissues. The blood sugar in untreated diabetic coma may be said to exist chiefly as alpha-beta glucose, a form which is not readily oxidized by living cells. Insulin brings about recovery by taking glucose from the blood, bringing about glycogen synthesis in the liver, which then breaks down to yield the more readily oxidizable form, namely gamma glucose.**

Benjamin P. Sandler, Lieutenant Commander (MC), U.S.N.R.

ANSWER.—There are several erroneous premises and conclusions in the foregoing:

1. It cannot be said that diabetic coma and hypoglycemic coma are both the result of the failure of the brain to burn glucose. For, while this is true in the latter condition, because of a failure of delivery of adequate amounts of glucose to the brain as a result of the low blood sugar levels, it has been shown (Himwich, H. E., and Nahum, L. H.: The Respiratory Quotient of the Brain, *Am. J. Physiol.* **101**:446 [Aug.] 1932) that in the total absence of insulin the brain is able to metabolize carbohydrates and maintain its respiratory quotient at 1.0. The wide differences in symptomatology of diabetic and hypoglycemic coma lend support to a difference in etiology of the two.

2. It is indeed doubtful that diabetic coma can develop in the absence of ketosis. The ketone bodies certainly cannot be excused from an important etiologic role in diabetic coma simply because they may be present at times without the occurrence of full blown diabetic coma. Their role in producing the disturbed acid-base relationship, acidosis and dehydration is well known. At least acetoacetic acid is believed to be toxic per se (Best, C. H., and Taylor, N. B.: The Physiological Basis of Medical Practice: A University of Toronto Text in Applied Physiology, ed. 3, Baltimore, Williams & Wilkins Company, 1943, p. 963). The depressed consciousness which may occur in the clinical syndrome we call diabetic coma is probably the result of several interdependent metabolic derangements, the sum of which, rather than any one singly, is responsible.

3. Ketosis apparently is not due to an inability of the tissues to oxidize glucose but rather seems to occur in conditions in which liver glycogen is seriously depleted (Best and Taylor: The Physiological Basis of Medical Practice, p. 1019. Peters, J. P.: A New Frame for Metabolism, *Am. Scientist.* **31**:36 [Jan.] 1943). Thus it is a finding in starvation and in phlorhizin diabetes, in which liver glycogen drops very low and energy requirements must be supplied largely by utilization of fats. Furthermore, in the depancreatized animal large doses of glucose administered intravenously will decrease ketonemia and ketonuria concomitant with storage of glycogen in the liver in spite of the fact that the glucose probably is not being burned by the peripheral tissues (Mirsky, I. A.; Heiman, J. D., and Broh-Kahn, R. H.: The Antiketogenic Action of Glucose in the Absence of Insulin, *Am. J. Physiol.* **118**:290 [Feb.] 1937).

Ketosis is not a finding in hypoglycemia, except in rare instances, as liver glycogen is not depleted in this condition. It is a rare possibility in renal diabetes with a very low renal glucose threshold (resembling the phlorhizinized animal [Wierzuchowski, M.: Intermediary Carbohydrate Metabolism: Vital Action of Glucose in Phlorhizin Diabetes, *J. Biol. Chem.* **73**:445 (June) 1927]) and in von Gierke's disease. In the former condition, depletion of liver glycogen occurs before ketonuria and hypoglycemia appear, and in the latter the liver, though filled with glycogen, is functionally without glycogen, as the glycogen present cannot be utilized. Infiltration of the liver with fat in the latter condition is common in the glycogen-depleted liver.

4. The question whether insulin acts to produce an active, or gamma glucose is still completely hypothetical. As far as can be determined, there is no definite evidence on this point.

PERSISTENT URINARY OBSTRUCTION AND INFECTION

To the Editor:—A man aged 28 has been under treatment for urinary symptoms of about two and a half years' duration. The symptoms began about January 1943 while at sea, with sudden inability to void. He passed a urinary calculus, and this was followed by pyuria. Examination of the kidney, ureter and bladder revealed an additional renal calculus in the right inferior calix and a right ureteral dilatation plus a right hydronephrosis. Further studies in the next few months revealed a urethral stricture and chronic cystitis. He was treated with many cystoscopies and lavage plus several courses of sulfathiazole and sulfapyridine. His urgency, frequency and dysuria improved slightly. He was readmitted to a navy hospital November 1943 to May 1944. During this stay he underwent cystoscopy; at this time films revealed contraction of the vesical neck, and a transurethral resection of the prostate was done. Following this he passed the previously mentioned stone, but considerable difficulty in urination remained. He became icteric, apparently as a result of sulfonamide medication. Liver function tests showed "considerable hepatic damage." The cystitis appeared to be due to Escherichia coli. Courses of penicillin, tyrothricin lavage and mandelic acid were of no avail. Urethral dilations have been done, but his urinary frequency (about every fifteen to thirty minutes) and severe tenesmus remain. The urine is full of white blood cells with scattered red blood cells; albumin is 2 plus. The blood cells appear normal. Several cultures of the urine showed hemolytic streptococci and nonhemolytic streptococci; others showed Alcaligenes fecalis. Is there anything further that can be done for this man? Of course the question of the newer drugs such as streptothricin has been brought up. Is it possible that one of these may be tried safely? It is felt that further courses of sulfonamides are inadvisable, since he has had severe reactions to them.

Jerome S. Davis, M.D., P. A. Surg., U. S. P. H. S. (R.).

ANSWER.—Although a bladder neck obstruction has been found and presumably removed the patient undoubtedly has further obstruction at that point as well as having an obstructing hydronephrosis; both factors are responsible for the persistence of this man's infection.

There does not seem to be any rationale to the continuation of all sorts of urinary antiseptics in a patient such as the one described. Thorough investigation of the cause of the obstruction should be made, with its relief, if possible, after which thorough prostatic and seminal vesicular massage should be given together with medicaments which would tend to keep the urine acid. Perhaps even this treatment will not be completely curative.

TINNITUS

To the Editor:—A white man aged 28, weighing 230 pounds (104 Kg.), 73 inches (185 cm.) tall, has had a constant moderately high pitched tinnitus in the right ear for the last six weeks. The drums and external auditory canals are normal except for a slight dilatation of the small blood vessel on the right drum running over the handle of the malleus. Both eustachian tubes are open and click on swallowing. Hearing does not seem to be impaired, but no audiometer tests have been done. A small amount of soft wax present was syringed out, but the tinnitus did not stop. The eyegrounds are normal and no scotomas, headaches, vertigo or vomiting have been present. Neurologic examination revealed no abnormalities. On clenching the teeth tightly the volume of the tinnitus is increased. X-ray of the teeth in the right upper jaw is normal, but several large cavities have been recently filled with silver. The blood count, urinalysis and blood pressure have been normal.

Oscar J. Balchum, M.D., Detroit.

ANSWER.—In the absence of other evident causes and in the presence of an apparently normal middle ear, as shown by the absence of important changes in the drum membrane, no satisfactory solution is possible in the problem presented without a careful testing of the hearing ability. The two most likely causes would be auditory nerve deafness and otosclerosis. Each can be diagnosed by tuning fork and audiometric examinations. There are cases of tinnitus in which no great loss of hearing is demonstrable on early examination. Only after a number of tests and the passage of some time can it be possible to assign the tinnitus definitely to a lesion in the hearing apparatus.

PASSAGE OF PENNY FROM STOMACH

To the Editor:—A boy aged 3 years has had a penny in his stomach for about two weeks. I have had him on a high residue diet, but the penny has not yet passed. The presence of the penny in the stomach does not seem to be causing him any inconvenience, but I am afraid that it may if it is allowed to remain over too long a time. At what age will the outlet of the stomach become adequate to allow the passage of the penny?

J. A. Orris, M.D., Windber, Pa.

ANSWER.—In all probability there is sufficient spasm of the outlet of the stomach to prevent the penny from passing. X-ray observations of stomachs of children at this age indicate that they are not so narrow as to prevent the passage of a penny. The patient might receive antispasmodics, particularly atropine, to allow the passage of the foreign body. Atropine in doses of 1/400 or 1/600 grain (0.16 or 0.1 mg.) administered three times daily might prove distinctly beneficial.

ANESTHETIC ACTION OF MAGNESIUM SULFATE

To the Editor:—It has been claimed that a 0.75 per cent solution of magnesium sulfate in water, injected around nerves or nerve roots, results in a prolonged anesthesia. 1. Is this claim well proved and generally accepted? 2. Could any possible harm result from the indiscriminate and wide use of 0.75 per cent magnesium sulfate solution as a solvent for the commonly used local anesthetics such as procaine and metycaine instead of water or saline solution?

Heinrich Lamm, M.D., La Feria, Texas.

ANSWER.—1. No.

2. S. J. Meltzer (*Berl. klin. Wchnschr.* **1**:73, 1906) experimented with this substance. He gave it subcutaneously, intravenously and intraspinally. He injected 1 cc. of a 25 per cent sterile solution of magnesium sulfate for each 12 Kg. of body weight and found that after three to four hours it was possible to operate painlessly on the legs and pelvic regions. He concluded that general anesthesia may often be produced by the intraspinal and subcutaneous injection of magnesium sulfate. From these experiments it appears that the use of a 0.75 per cent solution of magnesium sulfate as a solvent for the common local anesthetic agents might prolong the anesthesia, but the indiscriminate use of this solution would probably produce cerebral depression and a resultant general anesthesia.

Meltzer and Auer (*J. Exper. Med.* **123**:641, 1916) stated that the application of solutions of magnesium salts to nerve trunks interrupted conduction. A more or less complete block for afferent and efferent, for normal or artificial, impulses was established. This could be accomplished by hypertonic, isotonic and hypotonic solutions. The more concentrated the solution, the sooner the effect was established. In general, it takes more time for magnesium solutions to cause block of a nerve trunk than for other known local or general anesthetics.

DISINFECTION OF BARBERS' INSTRUMENTS AND STOCKINGS

To the Editor:—What solution or solutions should be used to sterilize barbers' instruments or other instruments that could be a means of transmittal of ringworm?

M.D., Iowa.

To the Editor:—Could you suggest a simple method of disinfecting stockings to destroy fungi—one that will not injure the stockings themselves? For your information, formaldehyde vapor removes the dye from nylon stockings.

M.D., California.

ANSWER.—1. The best means of sterilizing barbers' instruments is boiling them in sterile water. If this is not possible, they should be soaked in a 2 per cent cresol solution after being used on a person with ringworm of the scalp.

2. A dish of formaldehyde placed in a corner of a closed box and the stockings left in the box for twenty-four to forty-eight hours will generally kill the fungus. If the formaldehyde vapor removes the dye, the only thing that can be done is to wash the stockings repeatedly and thoroughly in soap and water.

TINGLING OF SHOULDER AND CERVICAL RIB

To the Editor:—A man awakened with a stiff neck after sleeping in a constrained position with his head bent over to the left. Later that day he had a tingling sensation in the left subclavicular fossa, similar to a galvanic treatment, which later had a sensation of being faradized almost to the point of a tetanic reaction. There is no particular pain, but the sensation "stops" him momentarily. Use of the left arm in an extended position seems to bring it on. In sleeping on the right side the tingling is felt first and can be stopped by quickly lying on the left side. The shoulder cap and the chest have been x-rayed and nothing can be seen. The blood pressure is 185/110. The Wassermann reaction is negative for syphilis. The patient is otherwise in good physical condition. Is this a scalenus anticus syndrome?

M.D., Missouri.

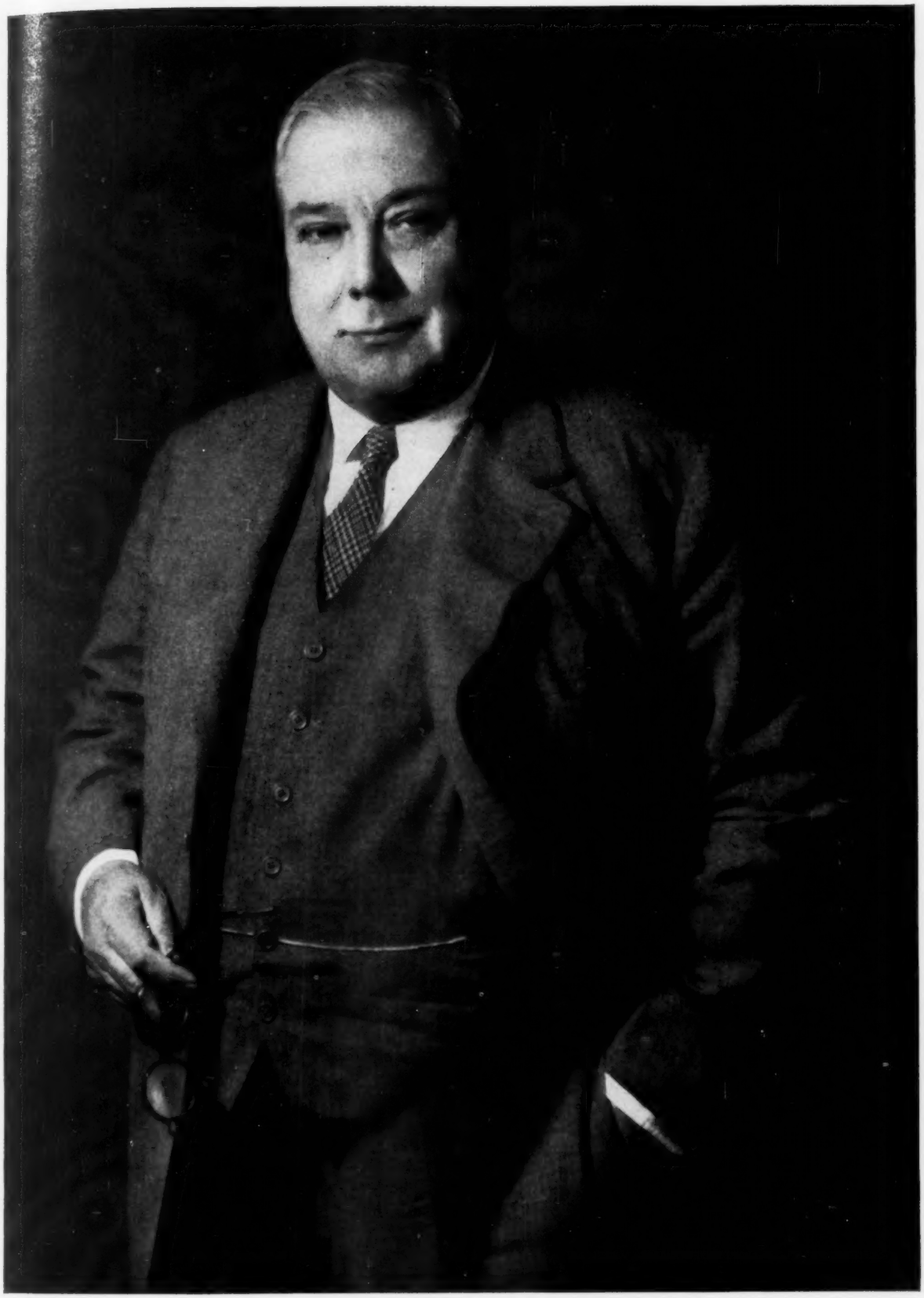
ANSWER.—Careful x-rays should be taken of the cervical spine to be certain that the patient does not have a cervical rib. If one is found, it will have to be decided whether or not it is causing the complaint. Cervical ribs do not always cause trouble. It could be a scalenus anticus syndrome but certainly of an unusual type. The patient's hypertension should be taken into consideration in treatment.

THYROID, METHYL TESTOSTERONE AND IMPOTENCE

To the Editor:—Has any improvement in cases of impotence ever been ascribed to thyroid? Has any loss of potency ever followed the use of methyl testosterone?

M.D., Illinois

ANSWER.—If impotence is associated with a thyroid deficiency it may be improved by the administration of desiccated thyroid. This form of treatment is not effective in the absence of a thyroid deficiency. Information on any loss of potency following the use of methyl testosterone has not been found.



Roger I. Lee

ROGER I. LEE, M.D.

PRESIDENT, AMERICAN MEDICAL ASSOCIATION, 1945-1946